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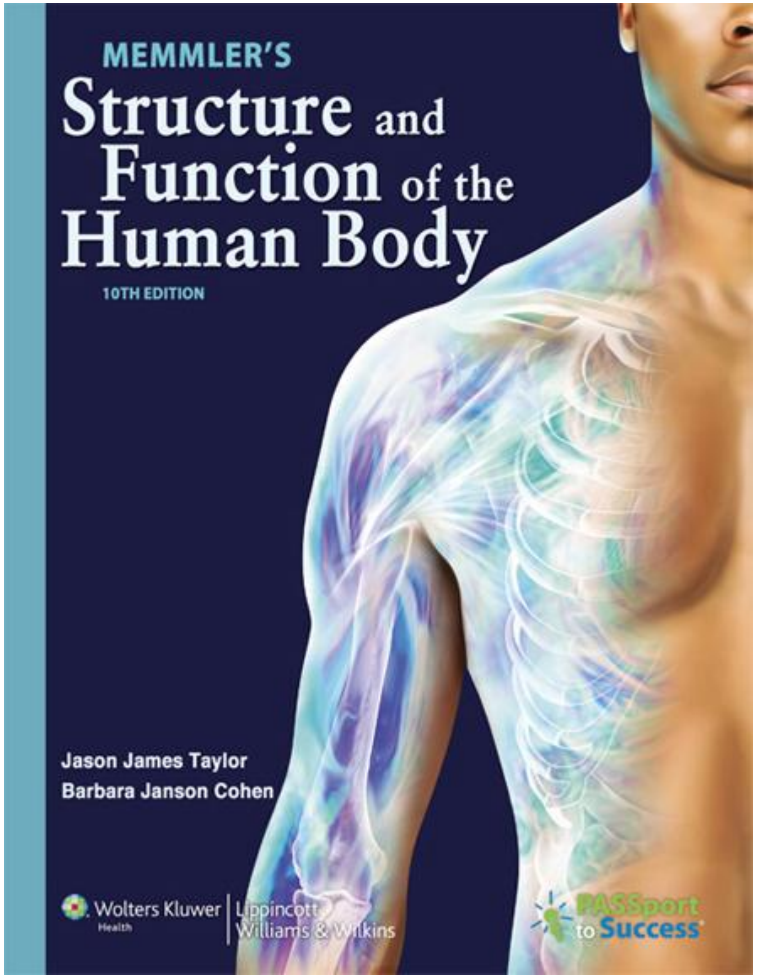

MEMMLER'S

Structure and Function of the Human Body

10TH EDITION

Jason James Taylor
Barbara Janson Cohen

Wolters Kluwer Health | Lippincott Williams & Wilkins

PASSport to Success

The Skin (Integumentary System)

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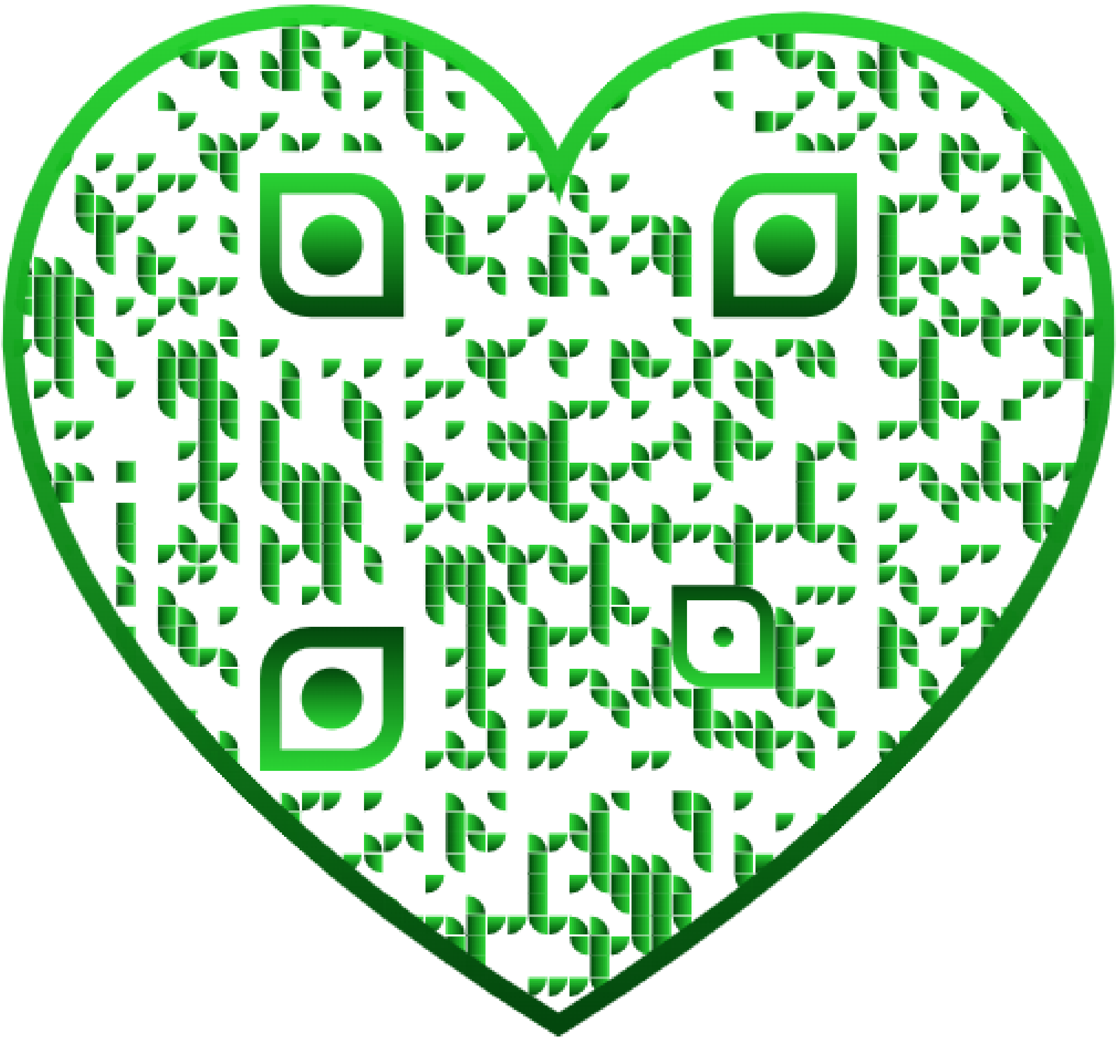

Dermatology

- **It is the branch of medicine dealing with**

  the skin, nails, hair and its diseases. It is a specialty

  with both medical and surgical aspects..

- **◉ The branch of medicine that deals with**

  diagnosis and treatment of diseases and disorders of

  skin. (oxford dic.)

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FACTS

- The skin is the largest organ of the body.
- ◉ Total area of about 15-20 square feet with thickness of 0.3 to 3mm.
- Accounts for 7% of body weight. It weighs twice as much as yourbrain.
- ◉ 1cm2 has 70 cm blood vessels, 55 cm nerves,100 sweat glands, 15 oil glands, 230 sensory receptors.

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Key Terms

apocrine
alopecia
arrector pili
cerumen
cicatrix
cyanosis
dermatitis
dermis
eccrine
epidermis
erythema
exfoliation
follicle
integument
jaundice
keloid
keratin
lesion
melanin
melanocyte
scar
sebaceous
sebum
stratum
subcutaneous
sudoriferous

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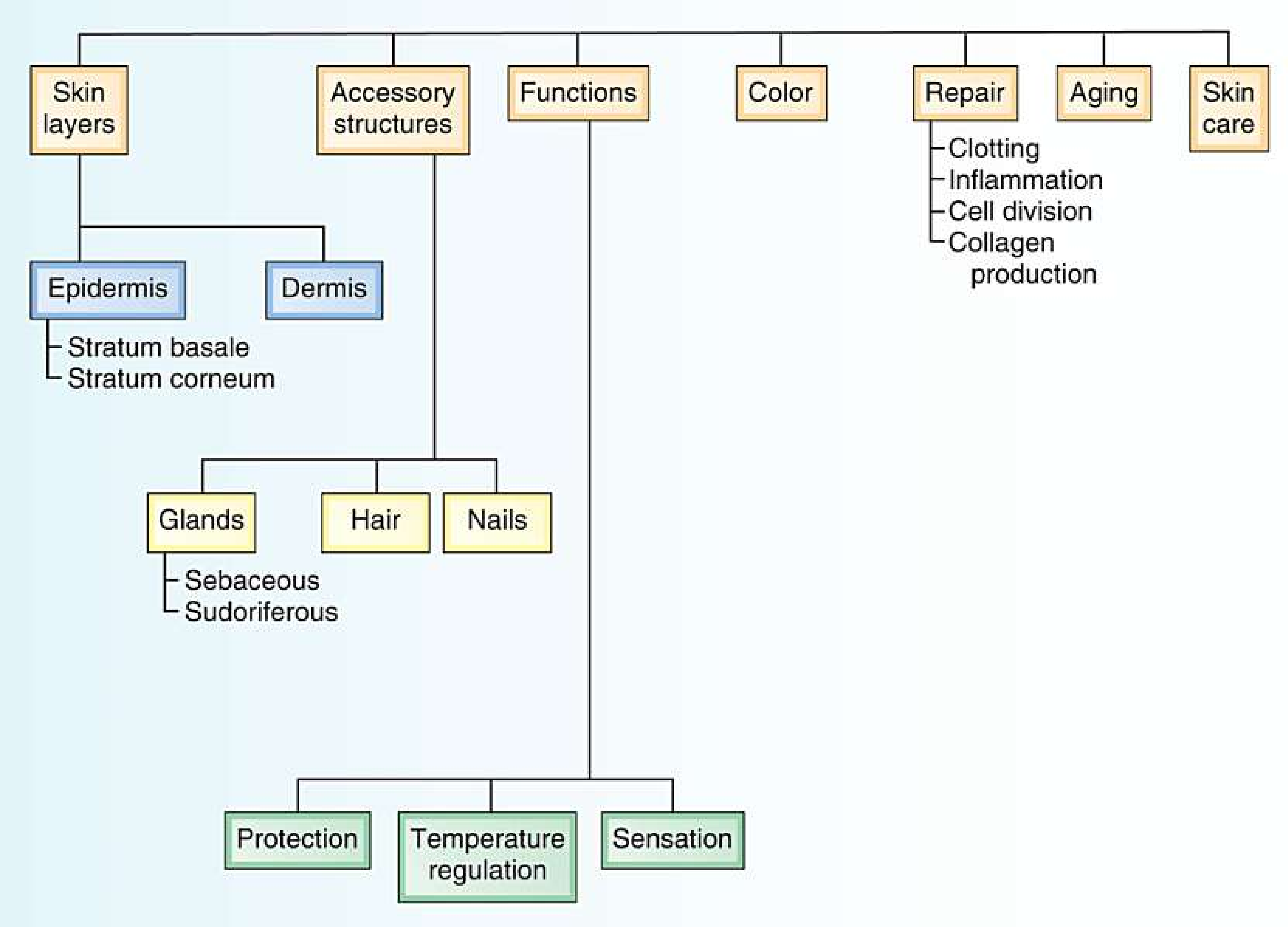
Overview



- Skin layers
  - Epidermis
    - Stratum basale
    - Stratum corneum
  - Dermis
- Accessory structures
  - Glands
    - Sebaceous
    - Sudoriferous
  - Hair
  - Nails
- Functions
  - Protection
  - Temperature regulation
  - Sensation
- Color
- Repair
  - Clotting
  - Inflammation
  - Cell division
  - Collagen production
- Aging
- Skin care

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Structure of the Skin

Learning Outcomes

1. Name and describe the layers of the skin.
2. Describe the subcutaneous layer.
3. Functions of skin.
4. Primary and secondary skin lesions.

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The Integumentary System

- Composed of two parts
  - Skin
  - Associated structures

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Structure of the Skin

- The skin consists of two layers
  - Epidermis
  - Dermis
- Underneath and supporting the dermis is the subcutaneous layer.

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Structure of the Skin

Epidermis

Location	Description	Function
Outermost portion of skin	- Composed mostly of stratified squamous epithelium - Avascular - Composed of several layers • **Stratum corneum** - Most superficial - Highly keratinized - Constantly shed • **Stratum basale** - Deepest - Produces new epithelial cells • **Stratum granulosum** • **Stratum spinosum**	- Protection from wear and tear, injury, and harmful substances - Melanin protects from UV radiation

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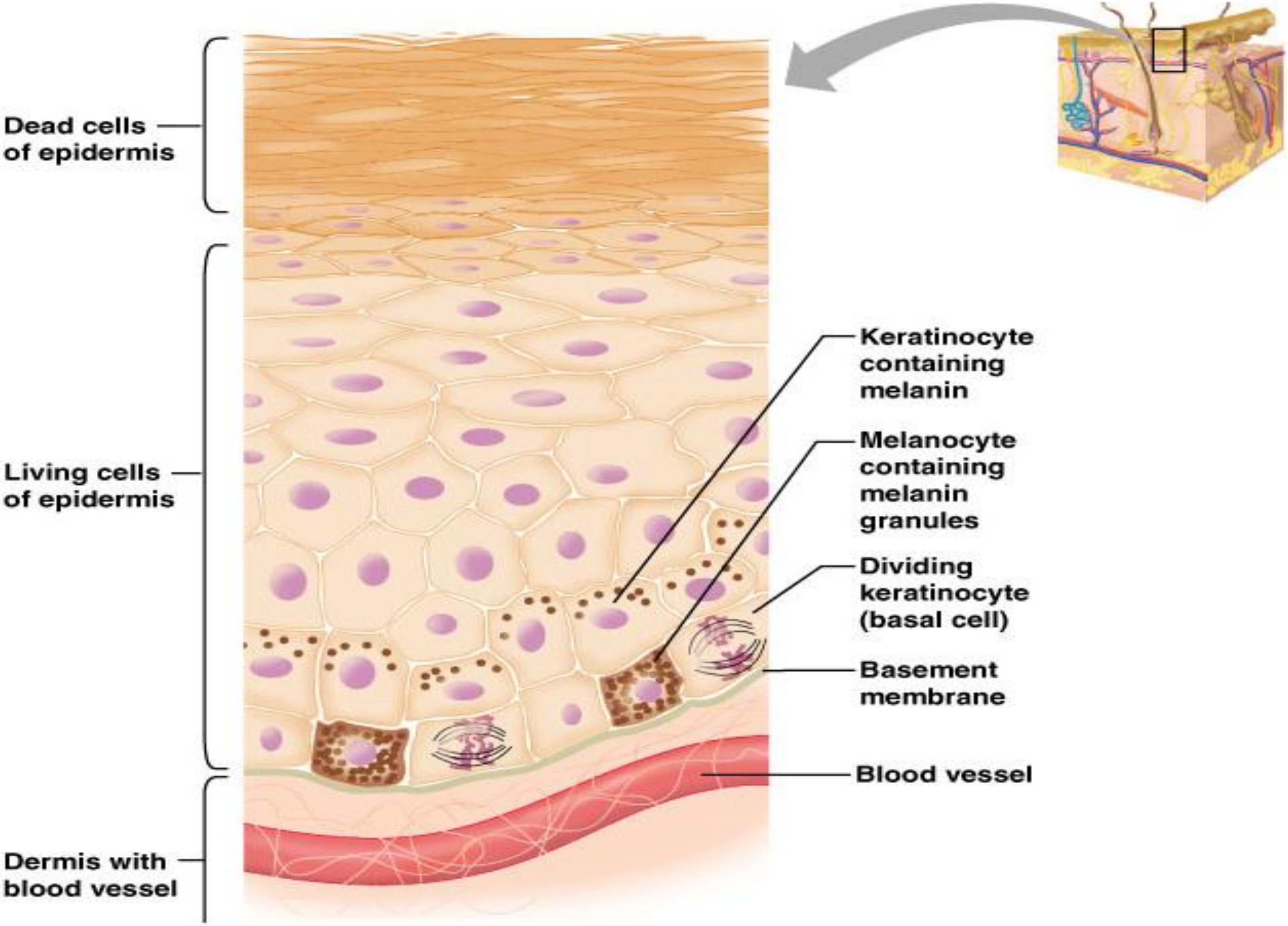
Dead cells of epidermis
Living cells of epidermis
Dermis with blood vessel
Keratinocyte containing melanin
Melanocyte containing melanin granules
Dividing keratinocyte (basal cell)
Basement membrane
Blood vessel

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Structure of the Skin

Dermis

Location	Description	Function
Beneath epidermis	- Composed of connective tissue - Vascular - Contains accessory structures • Hair follicles • Sebaceous glands • Sudoriferous glands • Sensory receptors • **Blood v**essels	- Protection - Nourishment of epidermis - Skin elasticity - Sensory perception

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Structure of the Skin

Subcutaneous Layer

Location	Description	Function
Beneath dermis	- Composed of loose connective tissue with large amounts of adipose tissue - Has blood vessels and nerve endings	- Connects skin to underlying muscle - Insulation - Temperature regulation - Sensory perception

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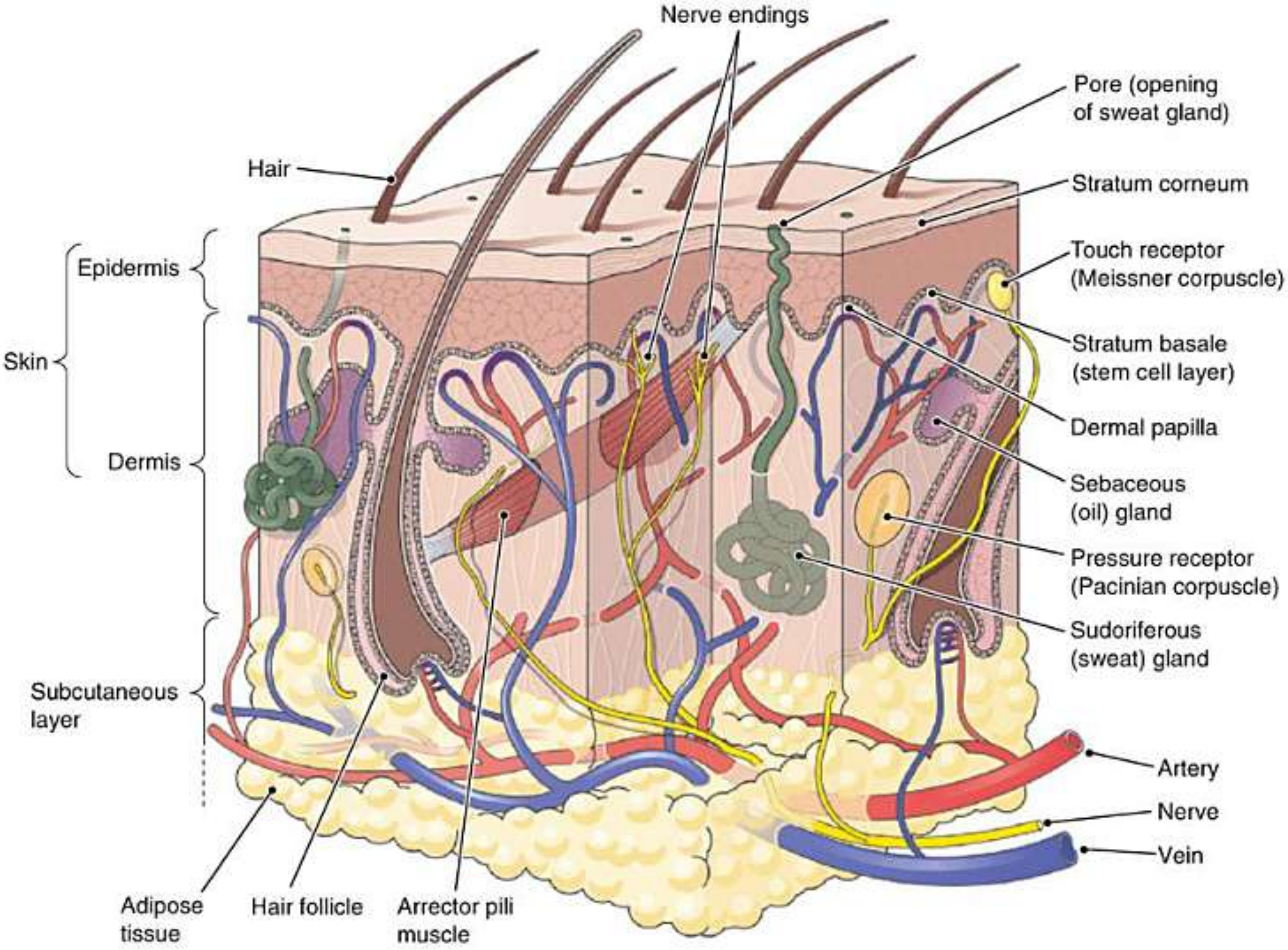
Figure 5-1 Cross-section of the skin.


Nerve endings
Hair
Pore (opening of sweat gland)
Stratum corneum
Epidermis
Skin
Dermis
Touch receptor (Meissner corpuscle)
Stratum basale (stem cell layer)
Dermal papilla
Sebaceous (oil) gland
Pressure receptor (Pacinian corpuscle)
Sudoriferous (sweat) gland
Subcutaneous layer
Artery
Nerve
Vein
Adipose tissue
Hair follicle
Arrector pili muscle

How is the epidermis supplied with oxygen and nutrients?
What tissue is located beneath the skin?

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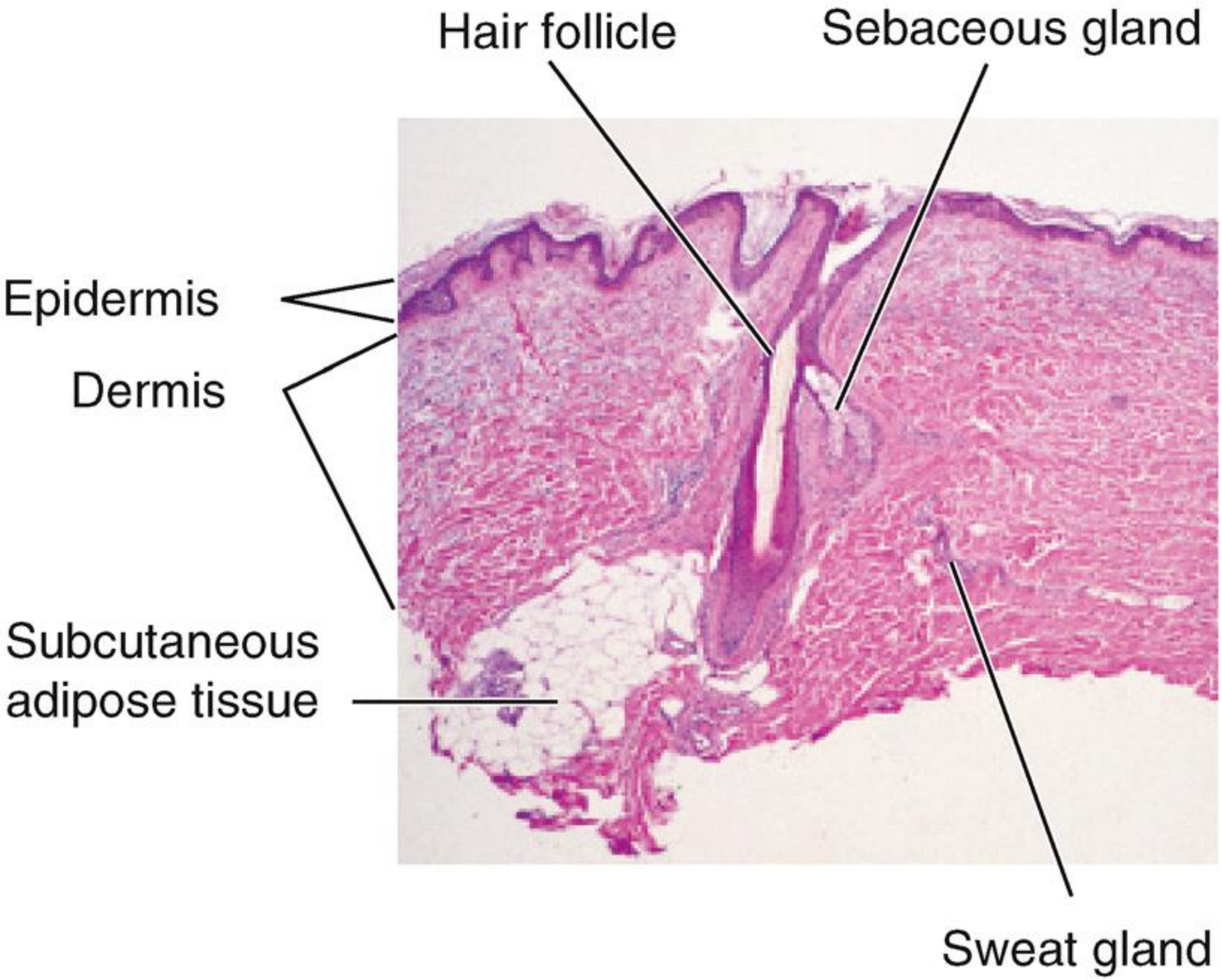
Figure 5-2 Microscopic view of thin skin.


Hair follicle
Sebaceous gland
Epidermis
Dermis
Subcutaneous adipose tissue
Sweat gland

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EPIDERMIS

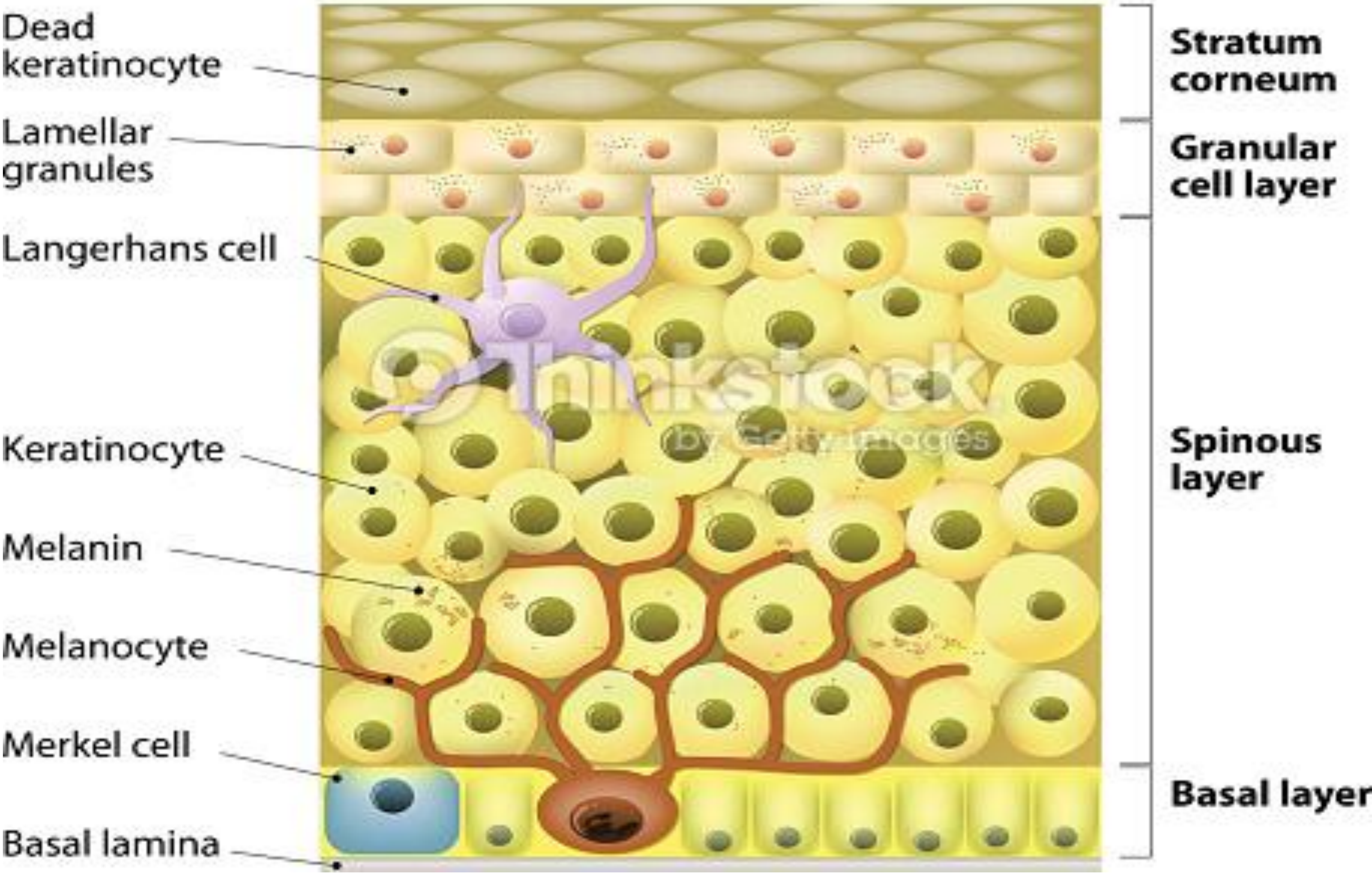
Dead keratinocyte
Lamellar granules
Langerhans cell
Keratinocyte
Melanin
Melanocyte
Merkel cell
Basal lamina
Stratum corneum
Granular cell layer
Spinous layer
Basal layer

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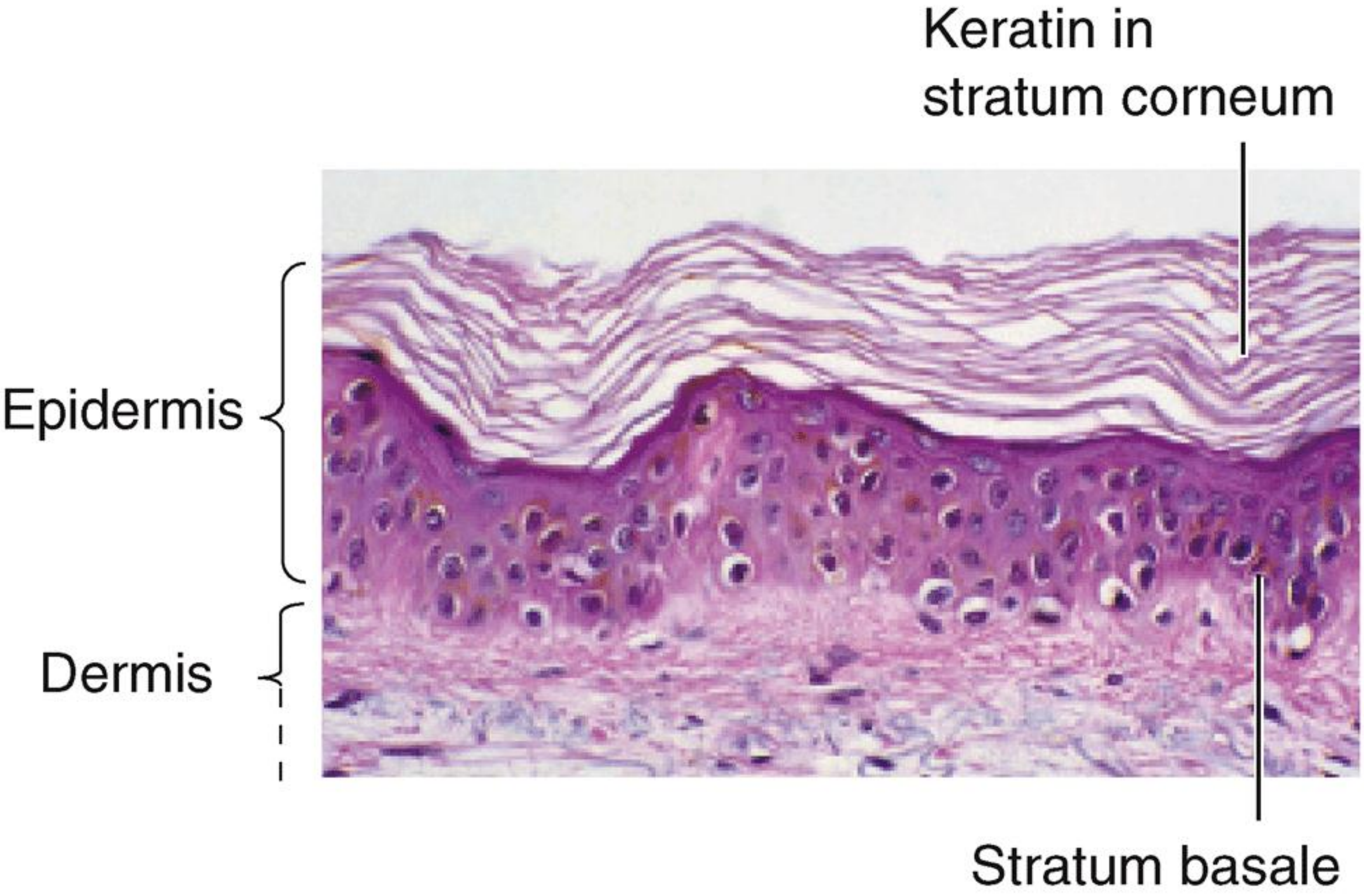
Figure 5-3 Upper portion of the skin.


Keratin in stratum corneum
Epidermis
Dermis
Stratum basale

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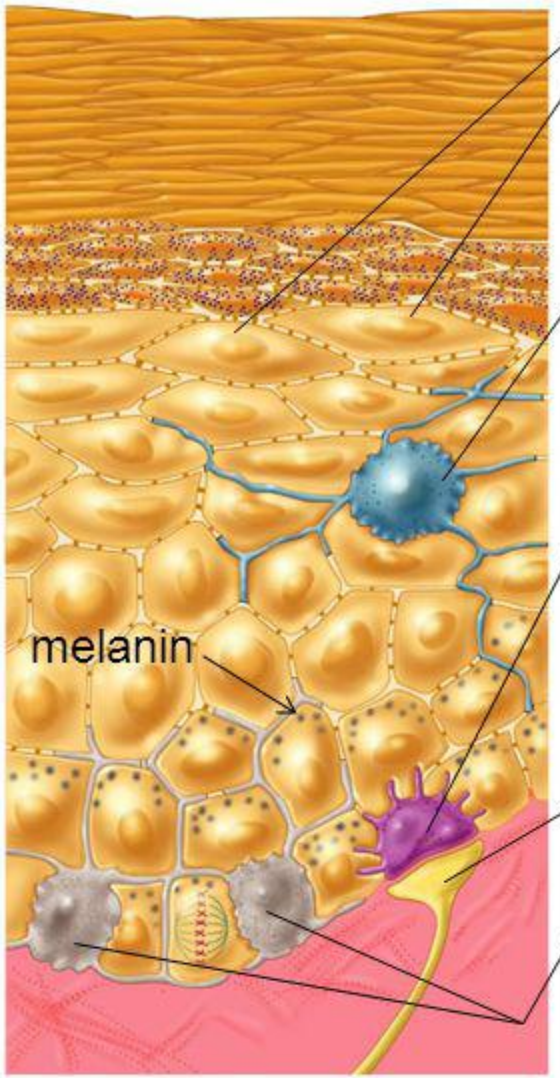
4 Epidermal Cell Types



Keratinocytes – make keratin fibrous protein

Langerhans' Cells – star shaped, epidermal dendritic phagocytic cells

Merkel Cells – half-sun touch receptors

- associated w/ **sensory nerve endings**

Melanocytes – makes brown pigment **melanin**

- shields keratinocyte DNA from UV damage

Three skin pigments

- Melanin
- Carotene: from carrots and yellow vegies
- Hemoglobin: the pink of light skin

◉ **Melanin** in granules passes from one melanocytes (same number in all races) to 36 keratinocytes via **melanosome.**

- Digested by lysosomes,
- Variations in color,
- Protection from UV light.

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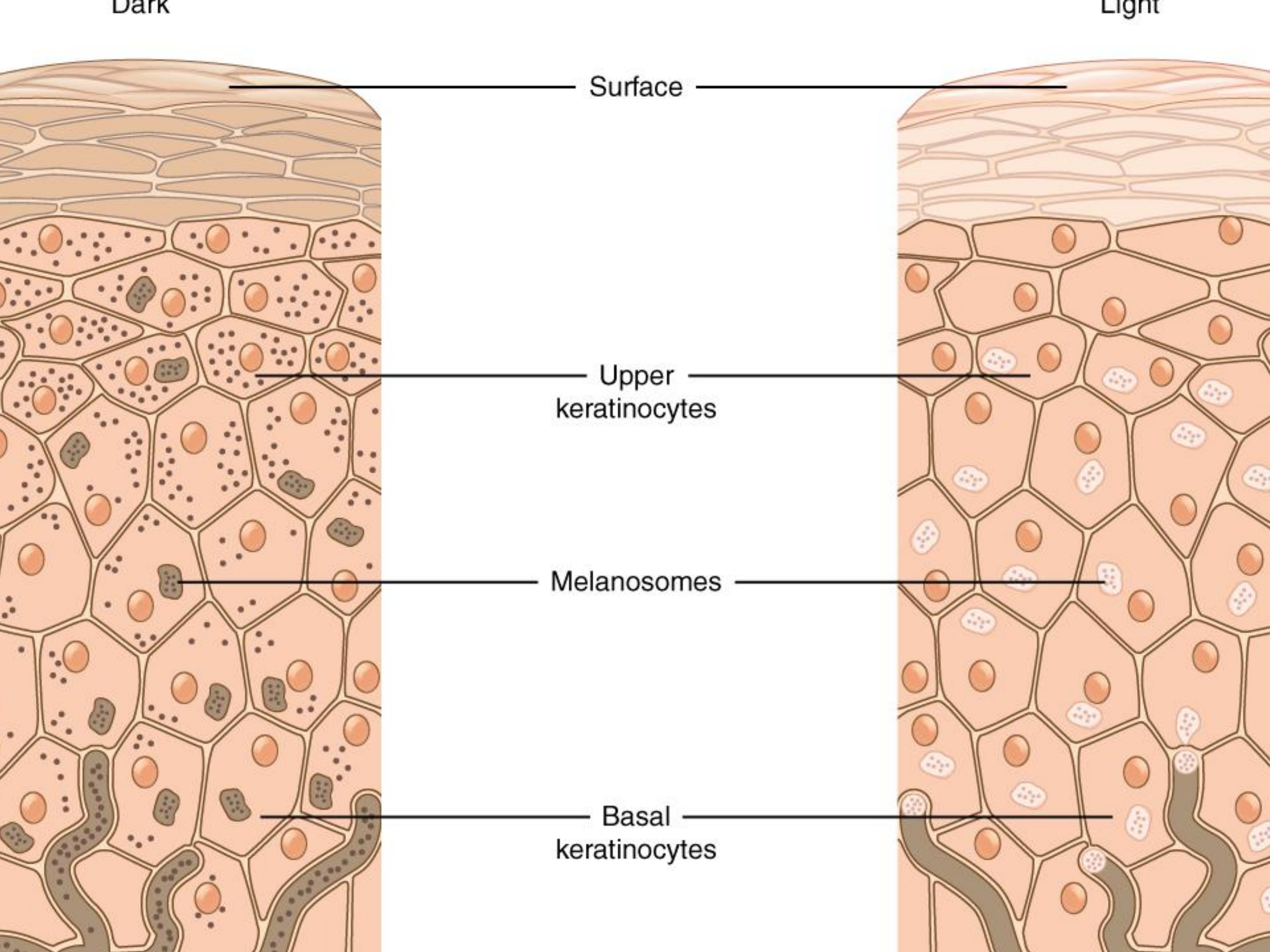
Dark
Light
Surface
Upper keratinocytes
Melanosomes
Basal keratinocytes

Structure of the Skin

Checkpoints

5-1 What is the name of the system that comprises the skin and all its associated structures?

5-2 Moving from the superficial to the deeper layer, what are the names of the two layers of the skin?

5-3 What is the composition of the subcutaneous layer?

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Structure of the Skin

Pop Quiz

Where do new epidermal cells come from?

A) Subcutaneous layer

B) Stratum basale

C) Stratum corneum

D) Dermis

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Structure of the Skin

Pop Quiz Answer

Where do new epidermal cells come from?

A) Subcutaneous layer
B) Stratum basale
C) Stratum corneum
D) Dermis

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Accessory Structures of the Skin

Learning Outcome

3. Give the location and function of the accessory structures of the integumentary system.

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Accessory Structures of the Skin

- Help protect the skin and give it more functions
- Include:
    - Sebaceous oil glands
    - Sweet glands
    - Hair
    - Nails

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Accessory Structures of the Skin

Glands

Structure	Description	Function
Sebaceous (oil) glands	- Saclike glands associated with hair follicles	- Secrete sebum, an oily substance that lubricates and water proofs the skin
Sudoriferous (sweat) glands	- Coiled glands that vent directly to the skin surface or through hair follicles	- Release perspiration to cool body by evaporation - Eliminate some soluble wastes

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Accessory Structures of the Skin

Hair and Nails

Structure	Description	Function
Hair	- Composed of keratin - Develops in a follicle - Grows from base of follicle - Structure • Follicle • Shaft • Root • Arrector pili muscle	- Conserve heat when raised by arrector pili muscles - Stimulates secretion of sebum
Nails	- Cover distal end of fingers and toes - Composed of keratin - Grow from proximal end - Structure • Root • Plate • Lunula	- Protection - Help in grasping objects

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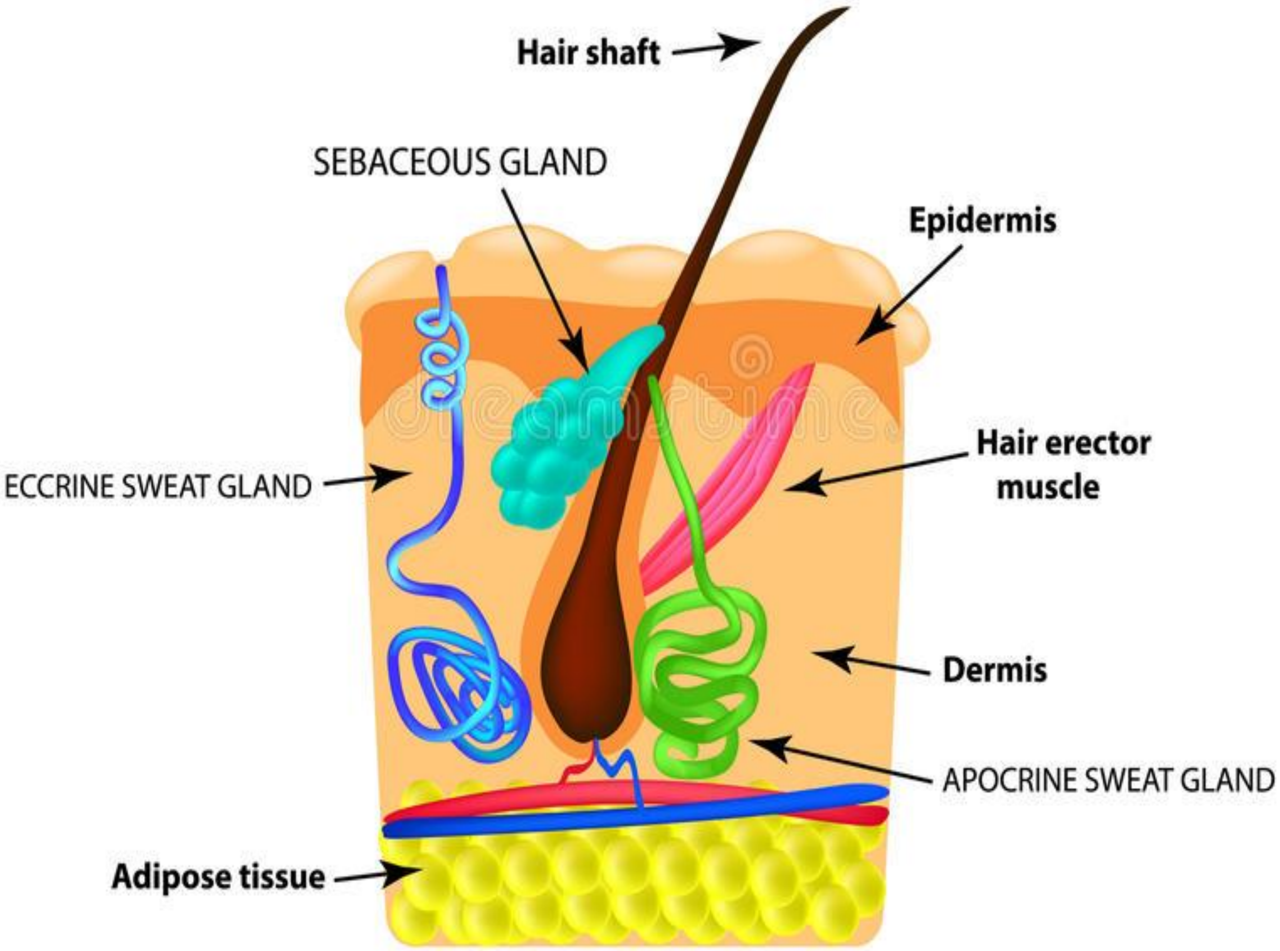
GLANDS OF THE SKIN


Hair shaft
SEBACEOUS GLAND
Epidermis
ECCRINE SWEAT GLAND
Hair erector muscle
Dermis
APOCRINE SWEAT GLAND
Adipose tissue

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- The sebaceous glands secrete an oily substance that
- protects the skin and prevent excess water loss.
- ◉ Meibomian glands **(eyelids)**
- ◉ Fordyce spots **(lips, gums and innercheeks,**
- **and genitals)**
- ◉ Areolar glands **(female nipples)**
- ◉ Several related medical conditions involve sebum
- —including
- ◉ acne, sebaceous cysts,
- ◉ hyperplasia, and sebaceous adenoma

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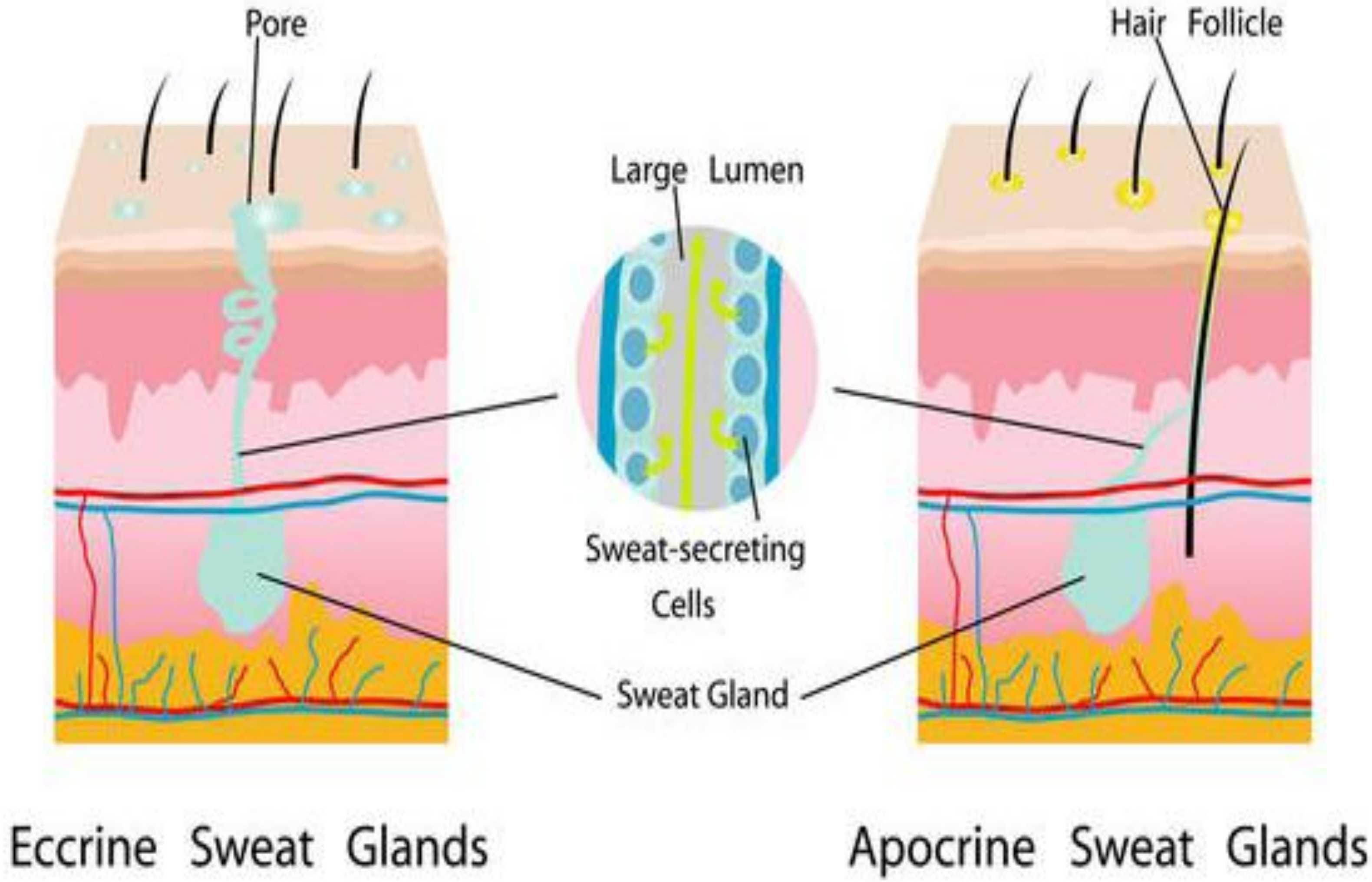
Pore
Hair Follicle
Large Lumen
Sweat-secreting Cells
Sweat Gland
Eccrine Sweat Glands
Apocrine Sweat Glands

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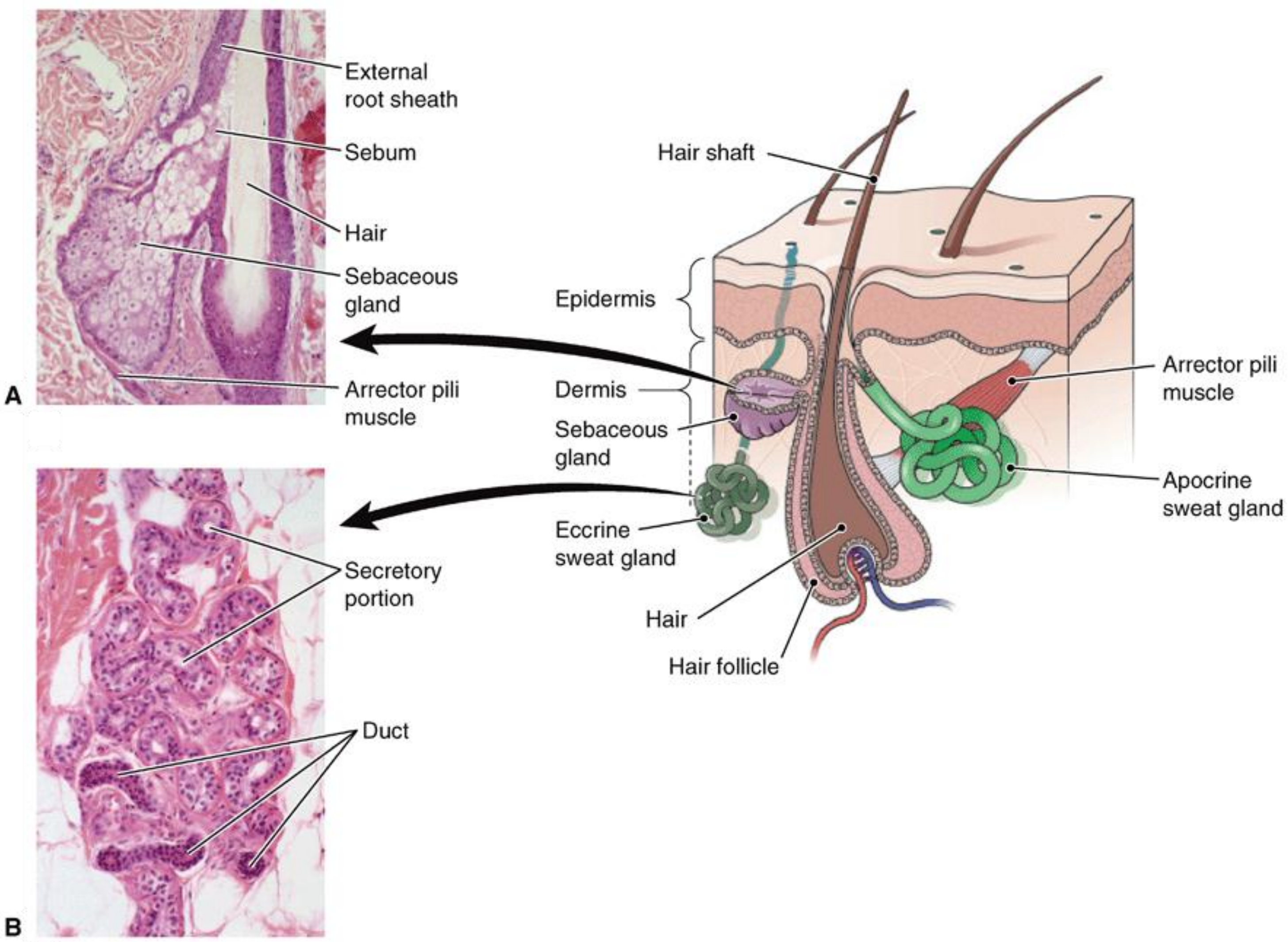
Figure 5-4 Portion of skin showing associated glands and hair.


External root sheath
Sebum
Hair
Sebaceous gland
Arrector pili muscle
A
Secretory portion
Duct
B
Hair shaft
Epidermis
Dermis
Sebaceous gland
Eccrine sweat gland
Hair
Hair follicle
Arrector pili muscle
Apocrine sweat gland

How do the sebaceous glands and apocrine sweat glands secrete to the outside?
What kind of epithelium makes up the sweat glands?

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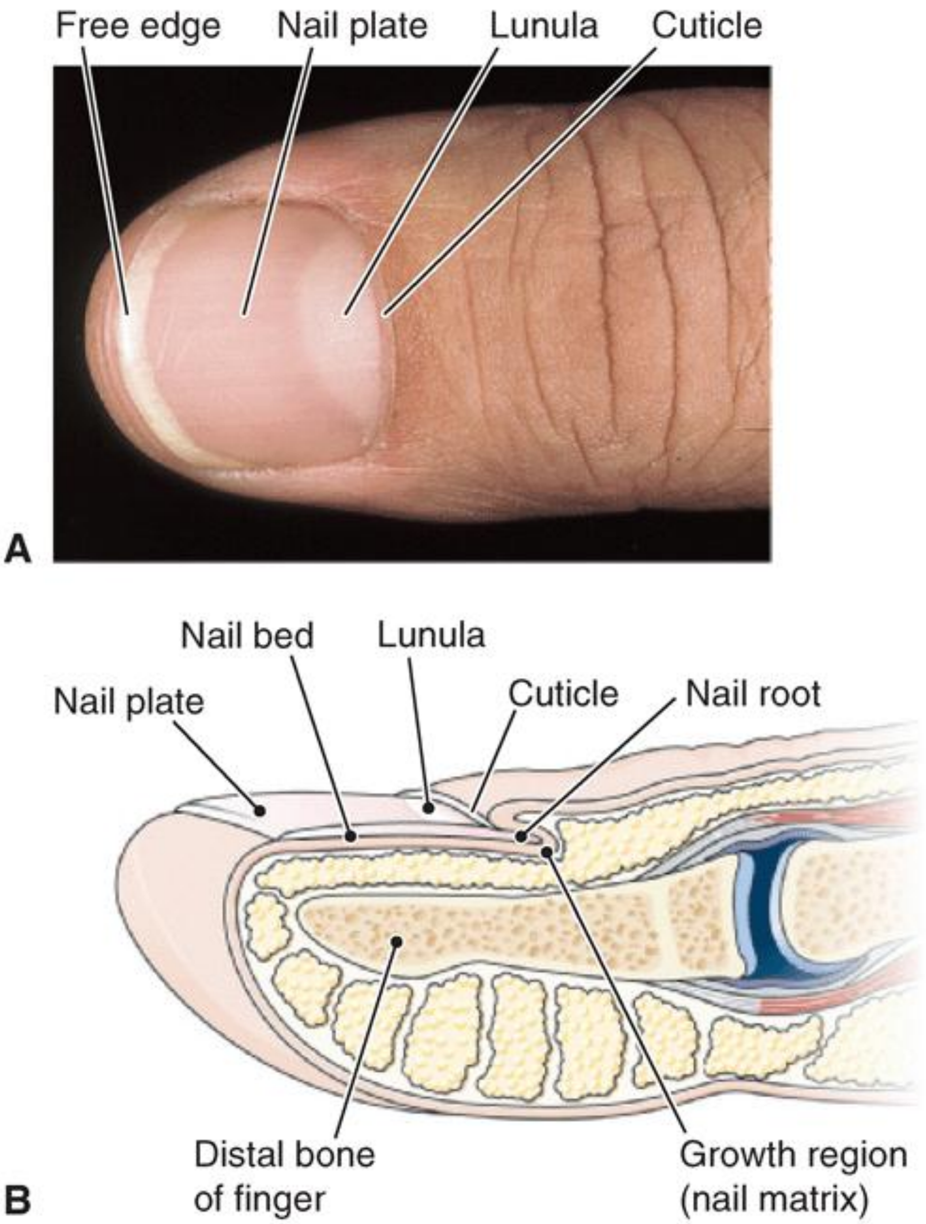
Figure 5-5 Nail structure.


Free edge
Nail plate
Lunula
Cuticle
A
Nail bed
Lunula
Nail plate
Cuticle
Nail root
Distal bone
of finger
Growth region
(nail matrix)
B

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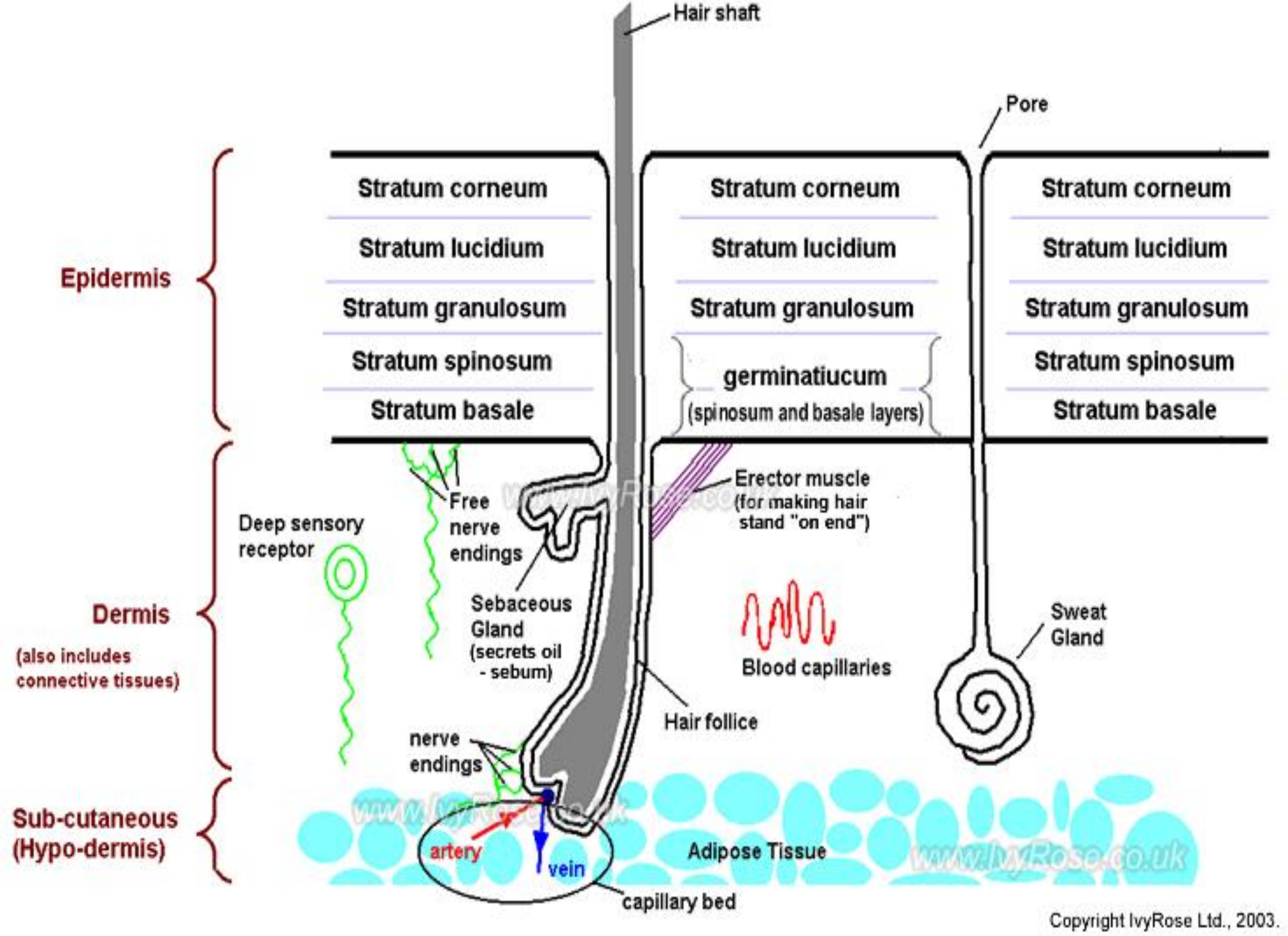
Hair shaft
Pore
Epidermis
Stratum corneum
Stratum lucidium
Stratum granulosum
Stratum spinosum
Stratum basale
germinatiucum
(spinosum and basale layers)
Dermis
(also includes connective tissues)
Deep sensory receptor
Free nerve endings
Sebaceous Gland (secrets oil - sebum)
Erector muscle (for making hair stand "on end")
Blood capillaries
Sweat Gland
Hair follice
nerve endings
Sub-cutaneous (Hypo-dermis)
artery
vein
Adipose Tissue
capillary bed
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Accessory Structures of the Skin

Checkpoints

5-4 What is the name of the skin glands that produce an oily secretion?

5-5 What is the scientific name for the sweat glands?

5-6 What is the name of the sheath in which a hair develops?

5-7 Where are the active cells that produce a nail located?

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Accessory Structures of the Skin

Pop Quiz

The maintenance of constant body temperature would be difficult without the actions of the

A) Apocrine glands

B) Meibomian glands

C) Sebaceous glands

D) Eccrine glands

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Accessory Structures of the Skin

Pop Quiz Answer

The maintenance of constant body temperature would be difficult without the actions of the

A) Apocrine glands

B) Meibomian glands

C) Sebaceous glands

D) Eccrine glasses

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Functions of the Integumentary System

Learning Outcome

4. List the main functions of the integumentary system.

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Functions of the Integumentary System

- Four major functions:
  1. Protection against infection
  2. Protection against dehydration (drying)
  3. Regulation of body temperature
  4. Collection of sensory information

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Functions of the Integumentary System

Protection Against Infection

- Intact skin forms a primary barrier against invasion.
- Interlocking pattern resists penetration.
- Shedding removes pathogens.
- Protects against bacterial toxins
- Protects against some harmful environmental chemicals

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Functions of the Integumentary System

Protection Against Dehydration

- Skin prevents water loss by evaporation.
  - Keratin in the epidermis
  - Sebum release from the sebaceous glands

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Functions of the Integumentary System

Regulation of Body Temperature

- Loss of excess heat and protection from cold are important functions of the skin.
  - Constriction of blood vessels
  - Dilation of blood vessels
  - Evaporation of perspiration

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Functions of the Integumentary System

Collection of Sensory Information

- Skin has many nerve endings and other special receptors.
  - Free nerve endings
  - Touch receptors (Meissner corpuscle)
  - Deep pressure receptors (Pacinian corpuscle)

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Functions of the Integumentary System

Other Activities of the Skin

- Absorption of substances such as medications
- Excretion
  - Water
  - Electrolytes
  - Wastes
- Manufacture of vitamin D

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Functions of the Integumentary System

Checkpoints

5-8 What two substances produced in the skin help to prevent dehydration?

5-9 What two mechanisms involving the skin are used to regulate temperature?

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Functions of the Integumentary System

Pop Quiz

Which of the following is NOT a function of skin?

A) Respiration

B) Excretion

C) Sensation

D) Thermoregulation

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Functions of the Integumentary System

Pop Quiz Answer

Which of the following is NOT a function of skin?

A) Respiration

B) Excretion

C) Sensation

D) Thermoregulation

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Color of the Skin

Learning Outcome

5. Discuss the factors that contribute to skin color.

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Color of the Skin

- Factors that influence skin color include:
    - Melanin
    - Hemoglobin
    - Carotene
    - Bile pigments

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Color of the Skin

Checkpoints

5-10 What are some pigments that impart color to the skin?

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Color of the Skin

Pop Quiz

Which pigment is responsible for a tan's brown color?

A) Melanin

B) Carotene

C) Hemoglobin

D) Bile

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Color of the Skin

Pop Quiz Answer

Which pigment is responsible for a tan's brown color?

A) Melanin

B) Carotene

C) Hemoglobin

D) Bile

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Repair of the Integument

Wound Healing

- Occurs only in areas with actively dividing cells
  - Epithelial tissues
  - Connective tissues
  - Minimally in muscle and nervous tissue

Factors That Affect Healing

  - Nutrition
  - Blood supply
  - Infection
  - Age

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Repair of the Integument

Checkpoint

5-11 What two categories of tissues repair themselves most easily?

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Word Anatomy

Word Part	Meaning	Example
derm/o	skin	The *epidermis* is the outermost layer of skin.
corne/o	cornified, keratinized	The *stratum corneum* is the outermost thickened, keratinized layer of the skin.
melan/o	dark, black	A *melanocyte* is a cell that produces the dark pigment melanin.
sub-	under, below	The *subcutaneous* layer is under the skin.
ap/o-	separation from, derivation from	The *apocrine* sweat glands release some cellular material in their secretions
pil/o	hair	The *arrector pili* muscle raises the hair to produce goose bumps.

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Description of Skin Lesions

Primary and ***secondary*** lesions (***eflorescence)***

- ✓ **single** or **multiple**
- ✓ **grouped** (multiple lesions grouped in one area)
- ✓ **disseminated** (exanthema – widespread discrete lesions)
- ✓ **arranged** (in line, in ring, snake-like…)

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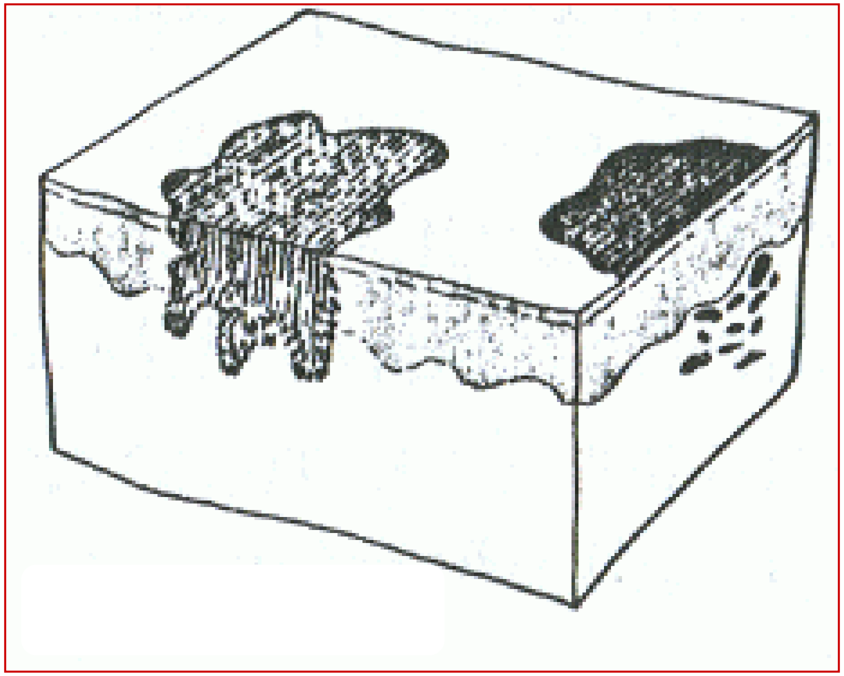
Primary Lesion



MACULE (spot)

any **flat** lesion, circumscribed discoloration, size < 1cm

PATCH = larger macule with some surface changes

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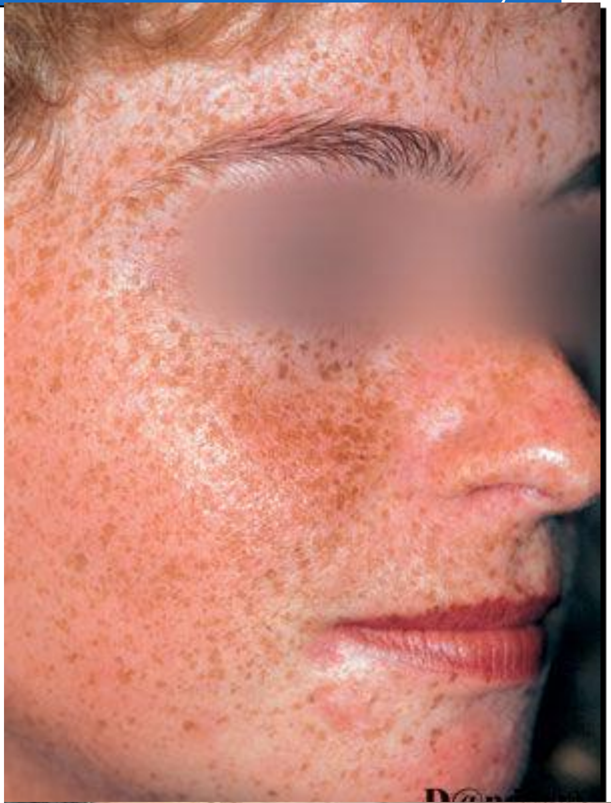
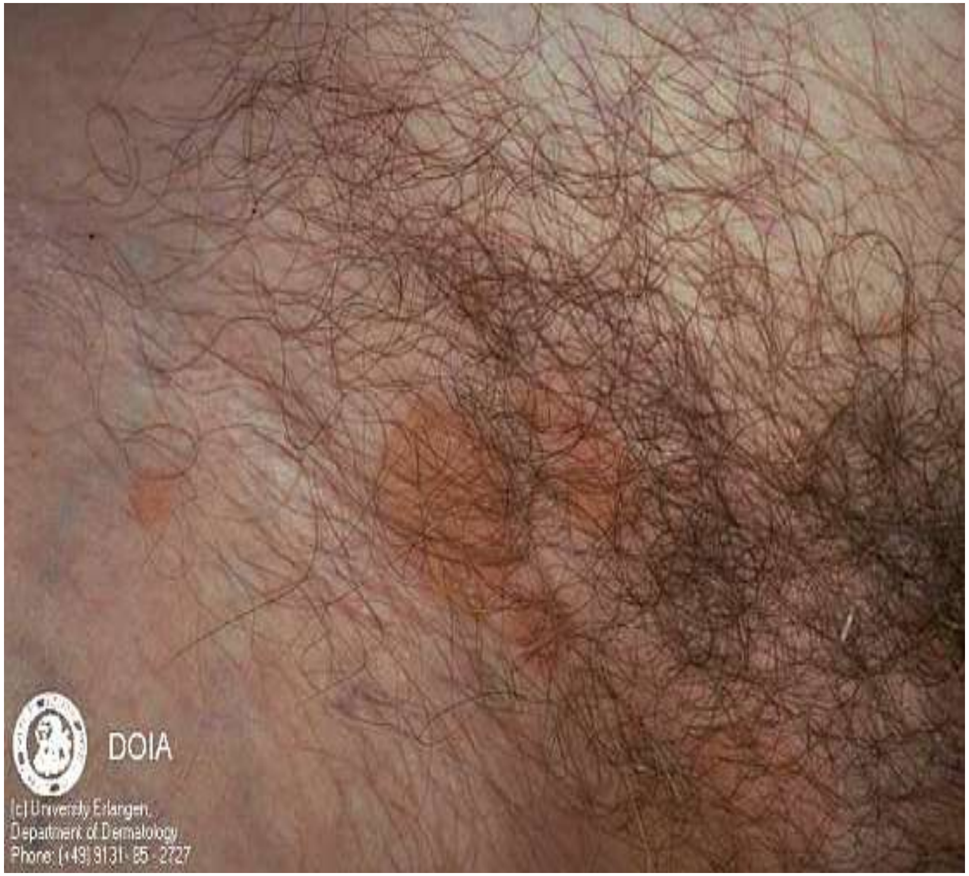
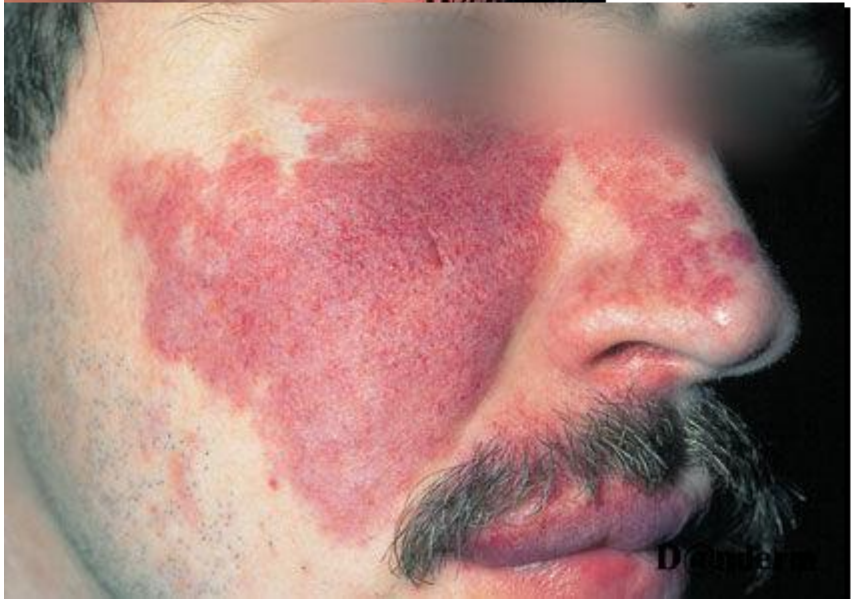
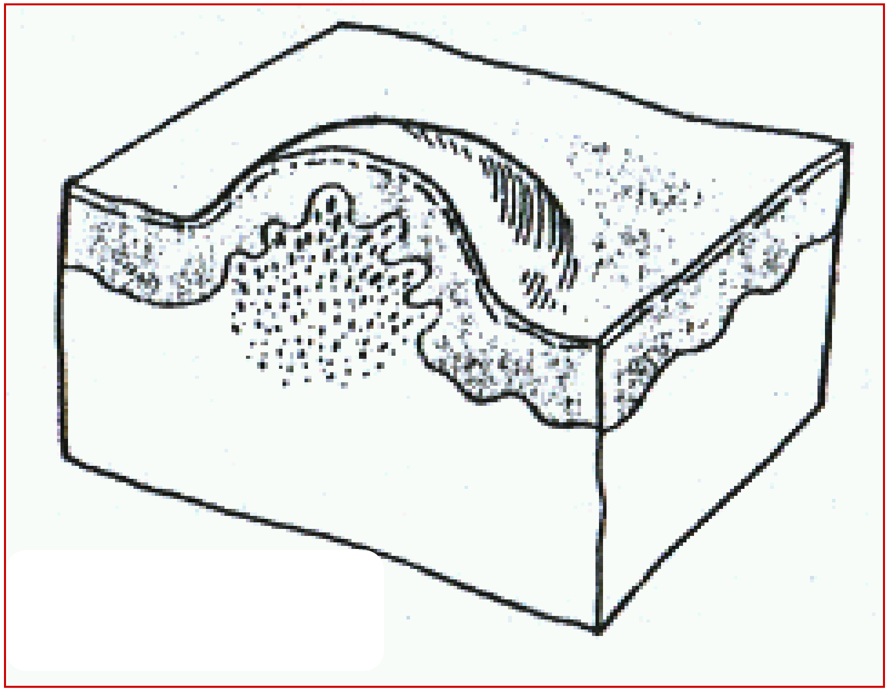
Patch & Macule




DOIA
(c) University Erlangen,
Department of Dermatology
Phone: (+49) 9131- 85 - 2727



Primary Lesion



PAPULE

any **elevated** lesion, size < 1cm, various color, papules may become confluent and form **plaques**

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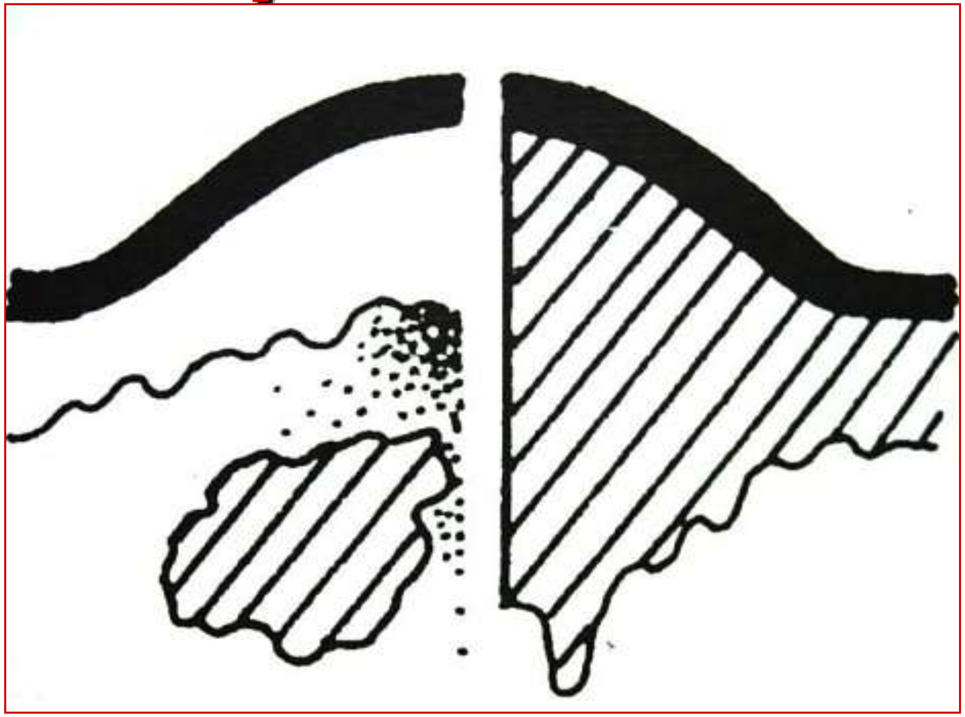
Primary Lesion



NODUL

any **elevated**, circumcribed lesion, **size > 1cm** in both diameter and depth

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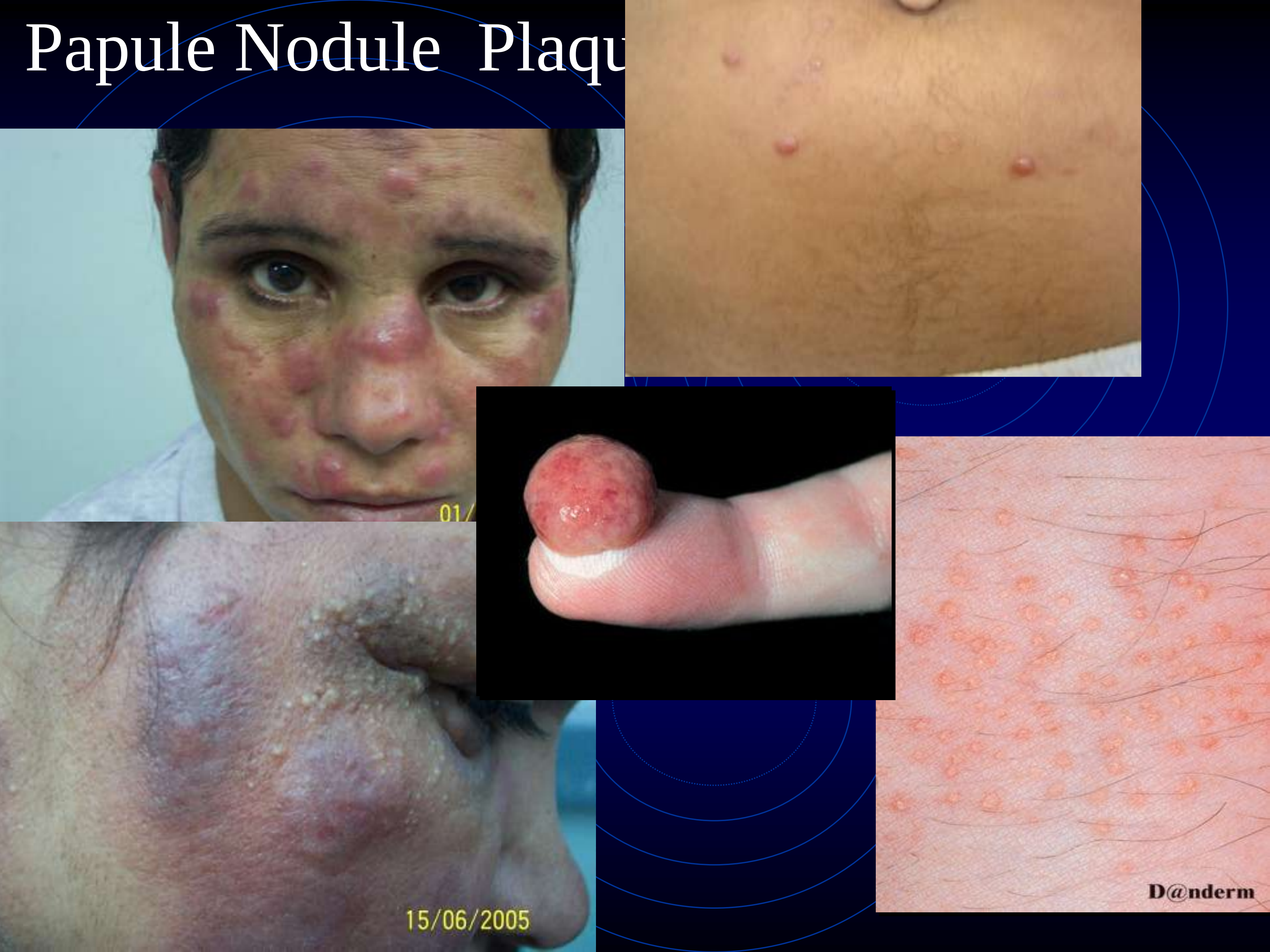
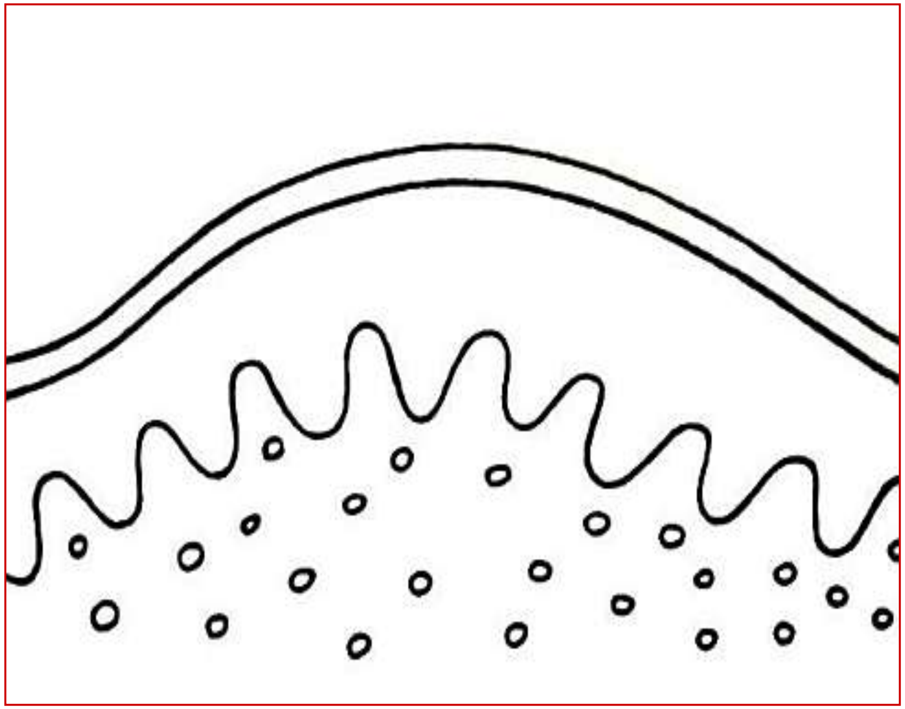
Papule Nodule Plaqu


01/
15/06/2005
D@nderm

Primary Lesion



URTICARIA (weal, hives, nettle-rash)

a firm plaque resulting from **dermal oedema**, it is **transient** - may last only **a few** hours

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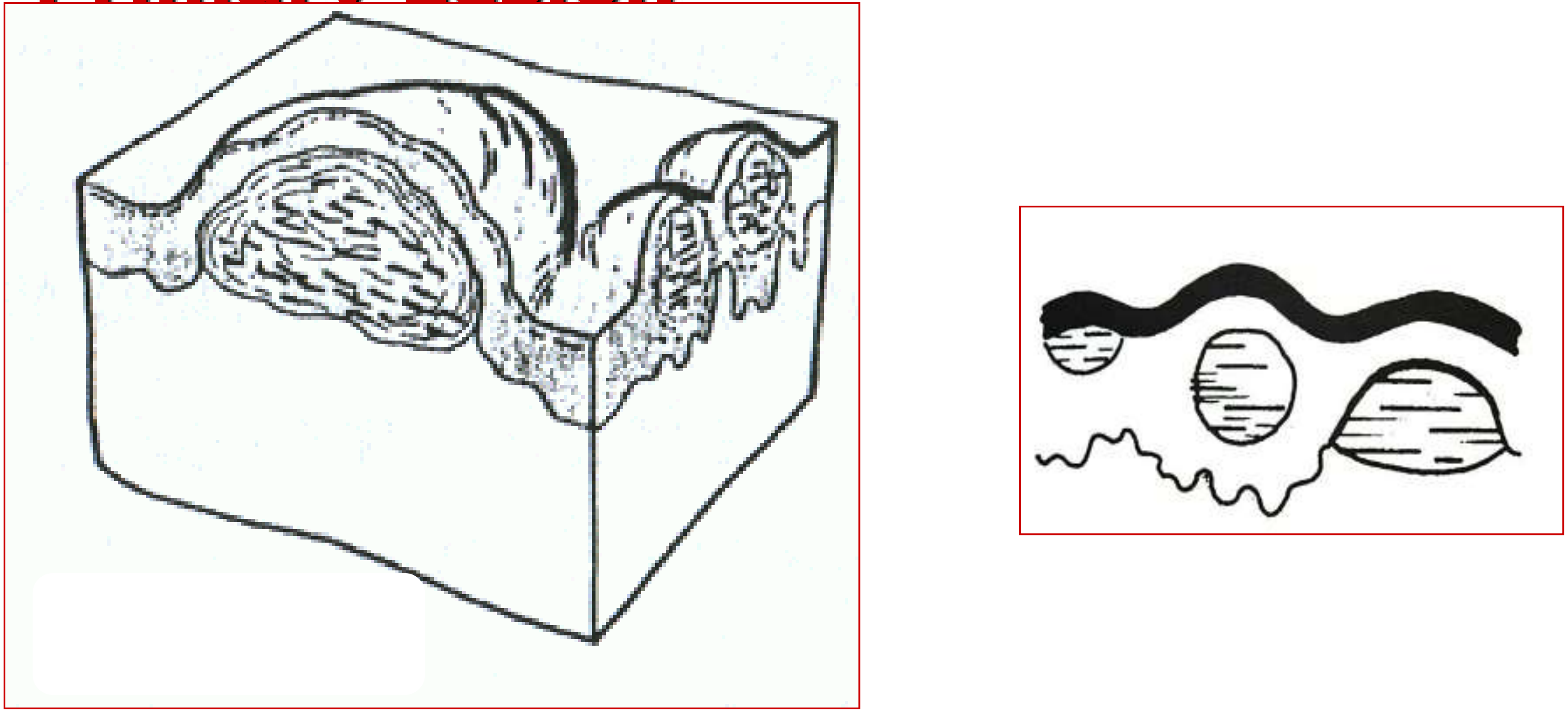
Primary Lesion



VESICLE (blister)

a fluid-filled lesion, size < 1cm,

BULLA - size > 1 cm in diameter

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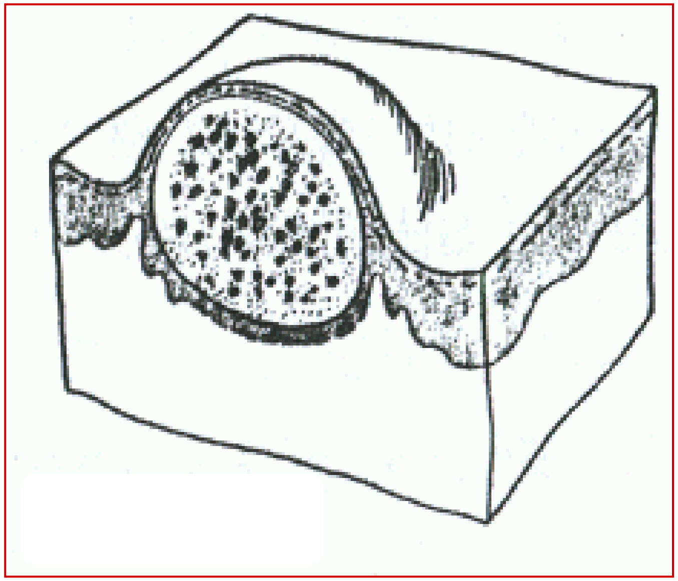
Primary Lesion



PUSTULE

a circumscribed collection of leukocytes and fluid, size < 1cm

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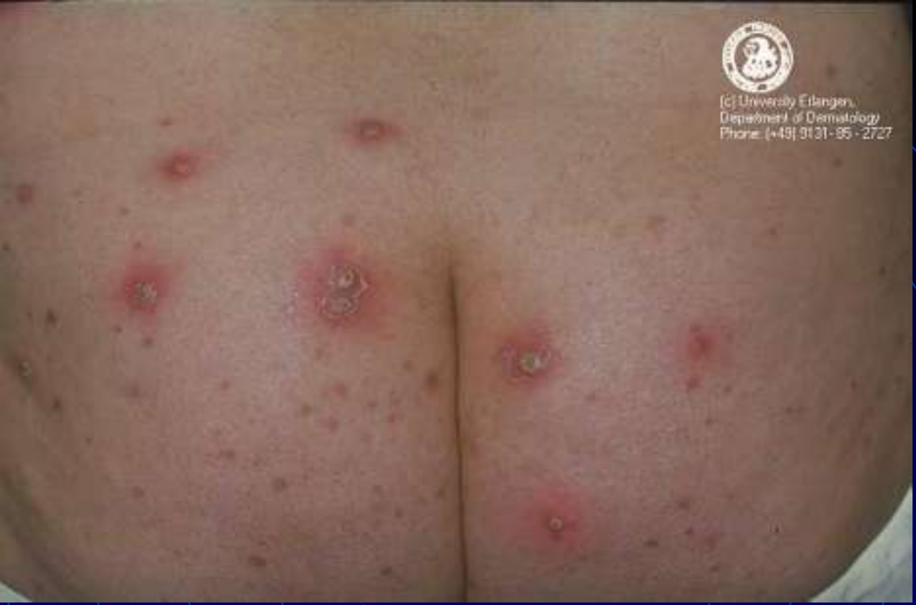

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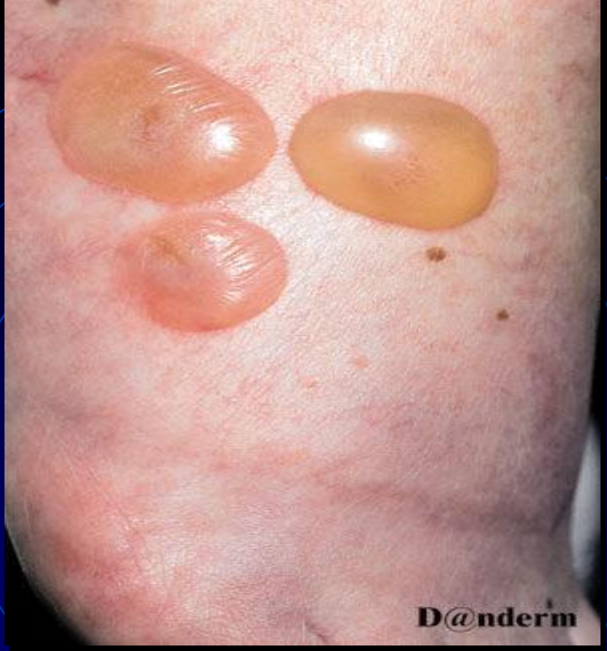
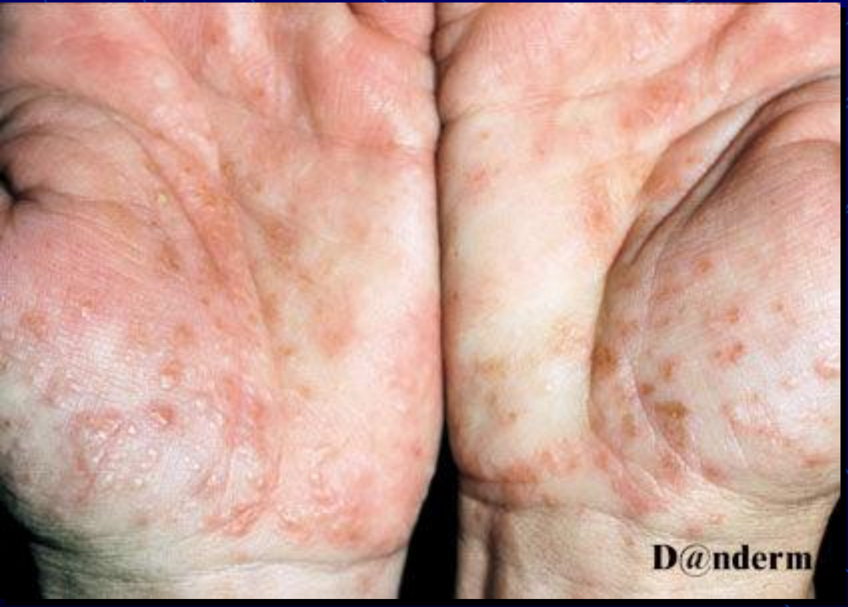
Department of Dermatology
Phone: (+49) 9131 - 85 - 2727



D@nderm



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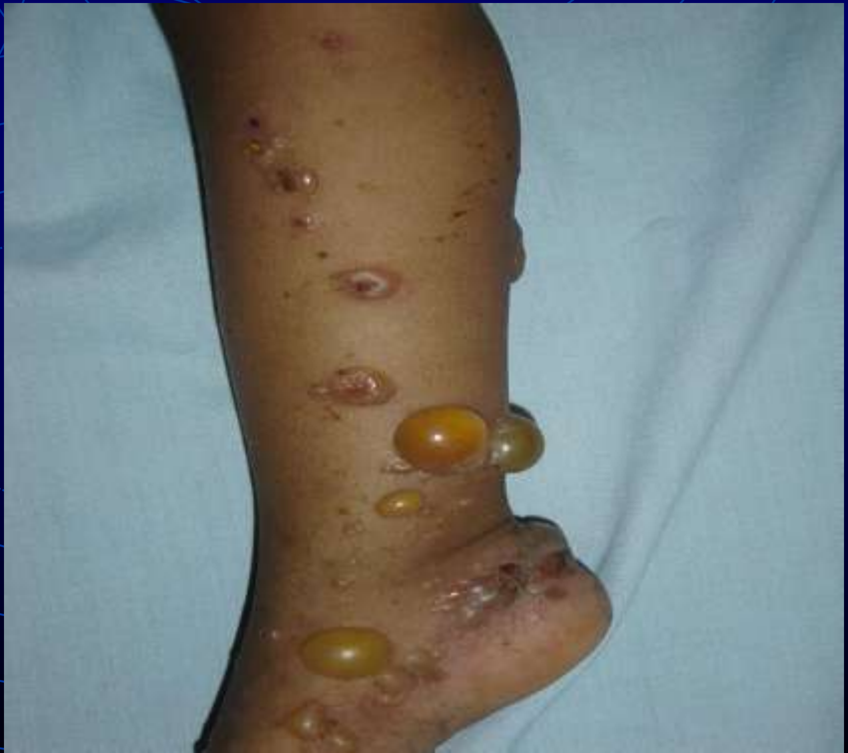

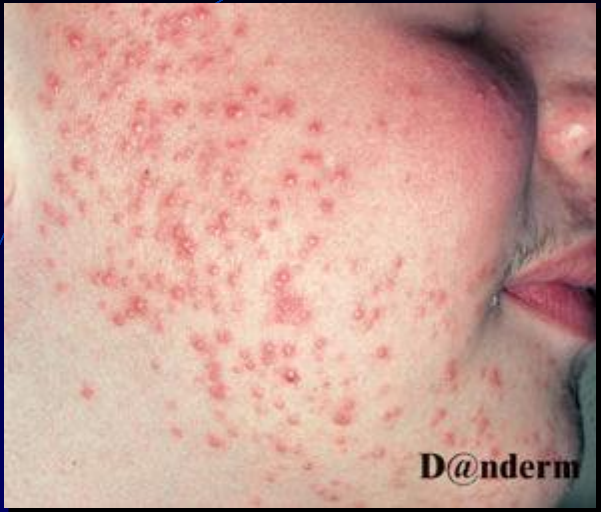
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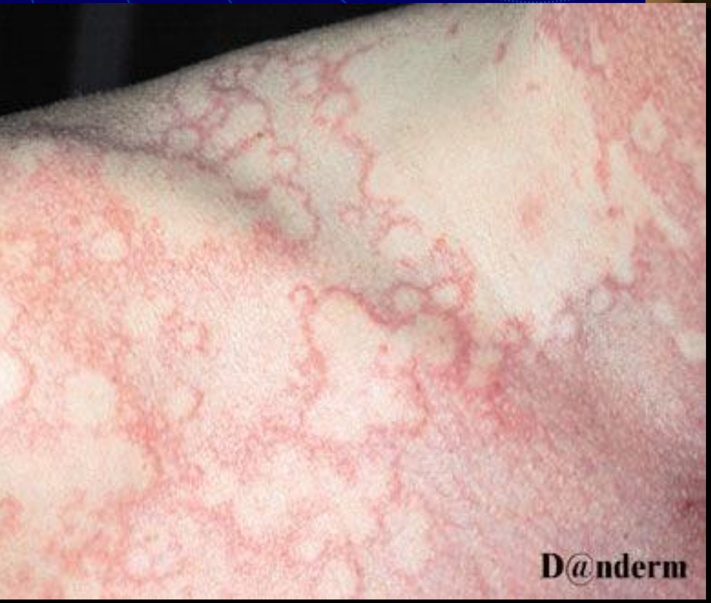
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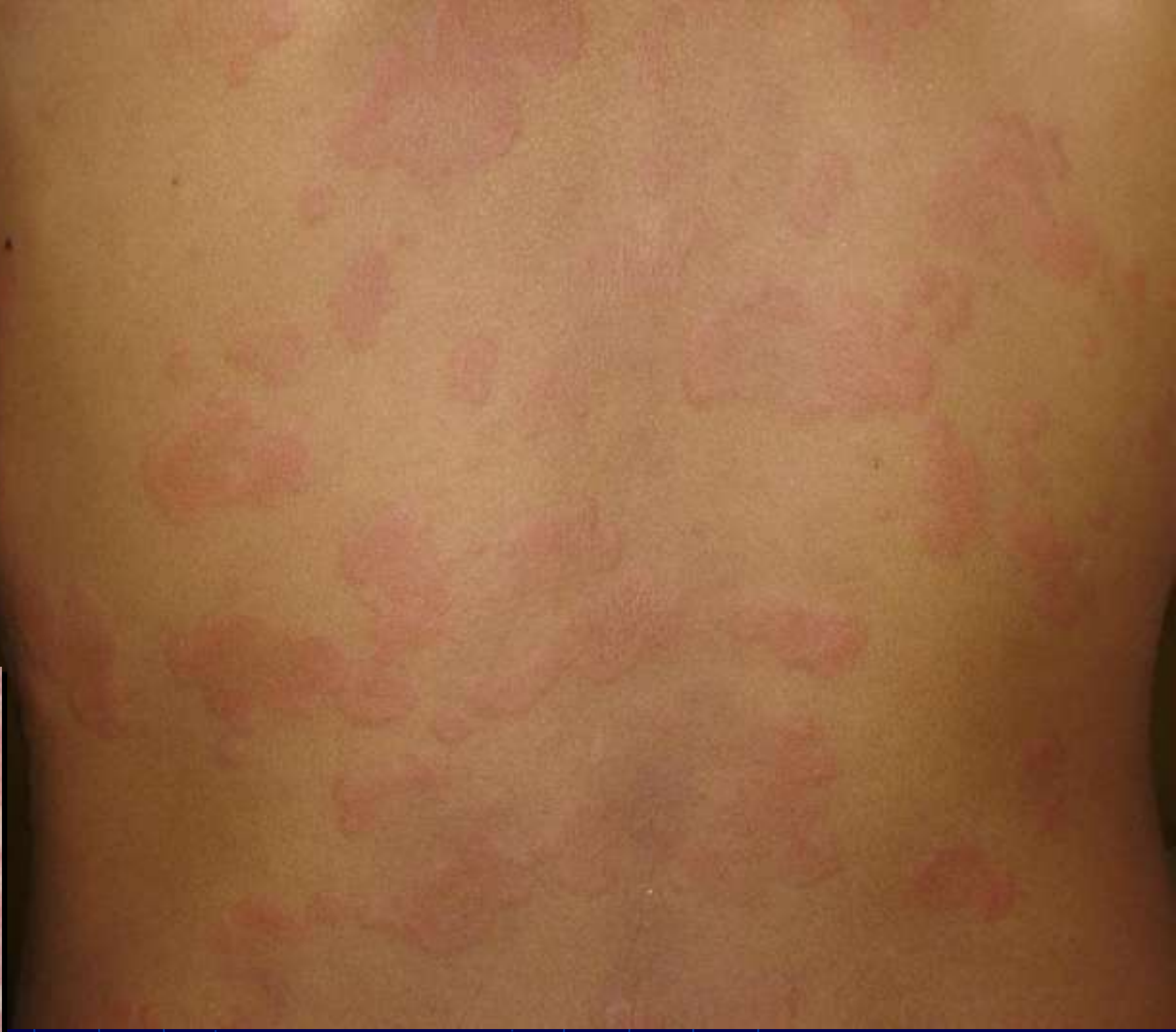
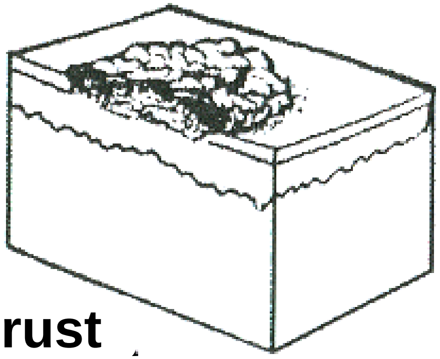
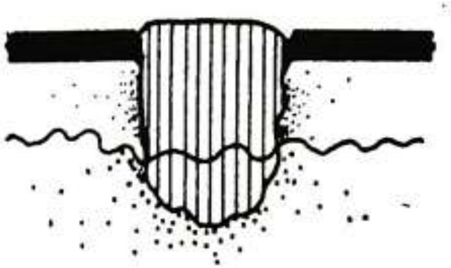


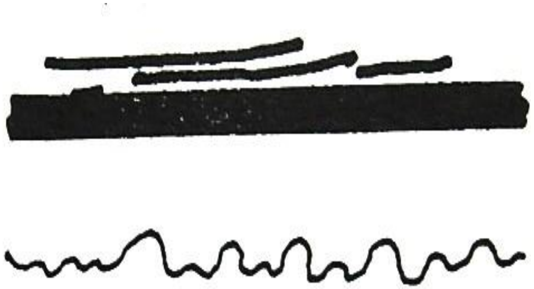
Secondary Lesions



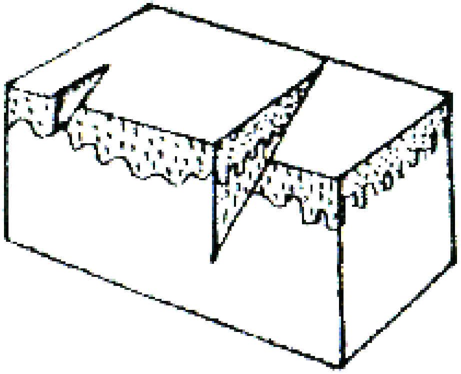
crust



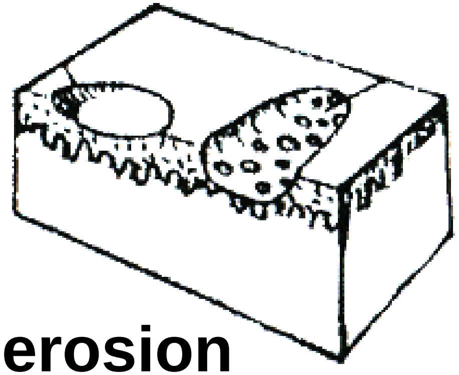
eschar (necrosis)



scale

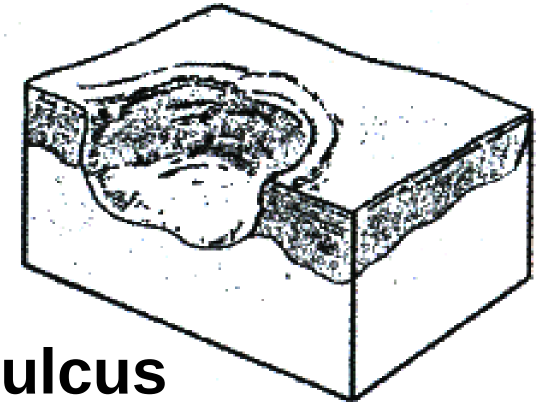


fissure



erosion

excoriation



ulcus

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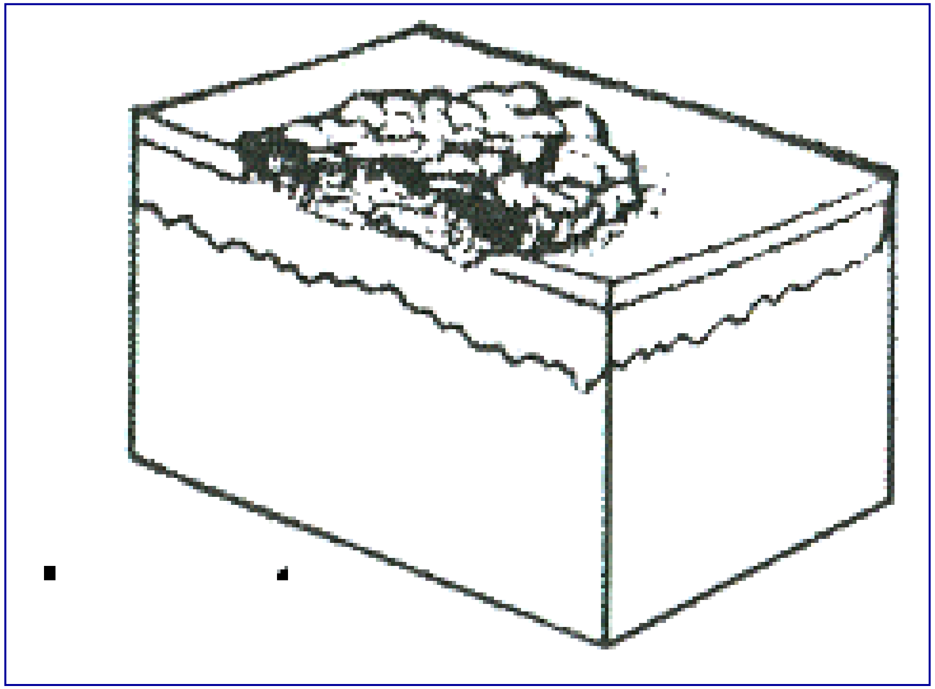
Secondary Lesion



CRUST

a collection of **dried** serum, blood or pus, and cellular debris

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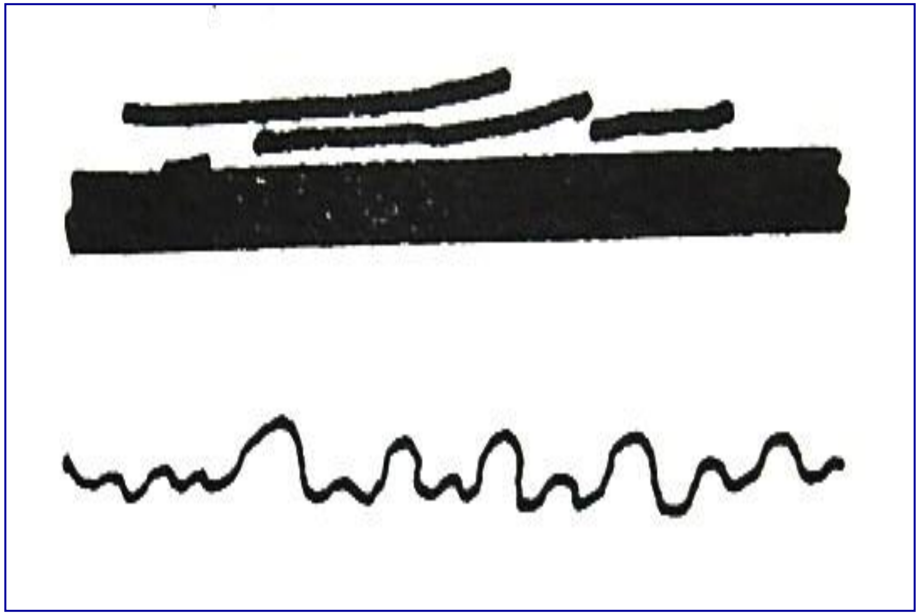
Secondary Lesion



SCALE (squama)

an excess **dead keratinocytes** that are produced by abnormal keratinization and shedding, scale are **dry** and usually whitish or yellowish

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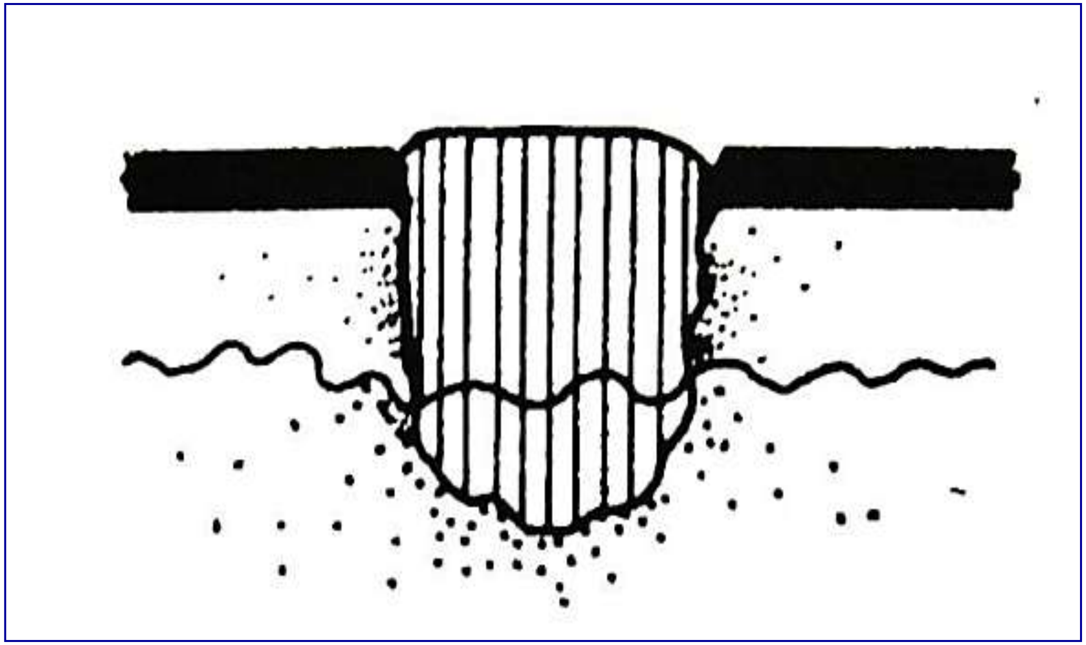
Secondary Lesion



ESCHAR **(necrosis)**

(e.g. bedsore, burn…)

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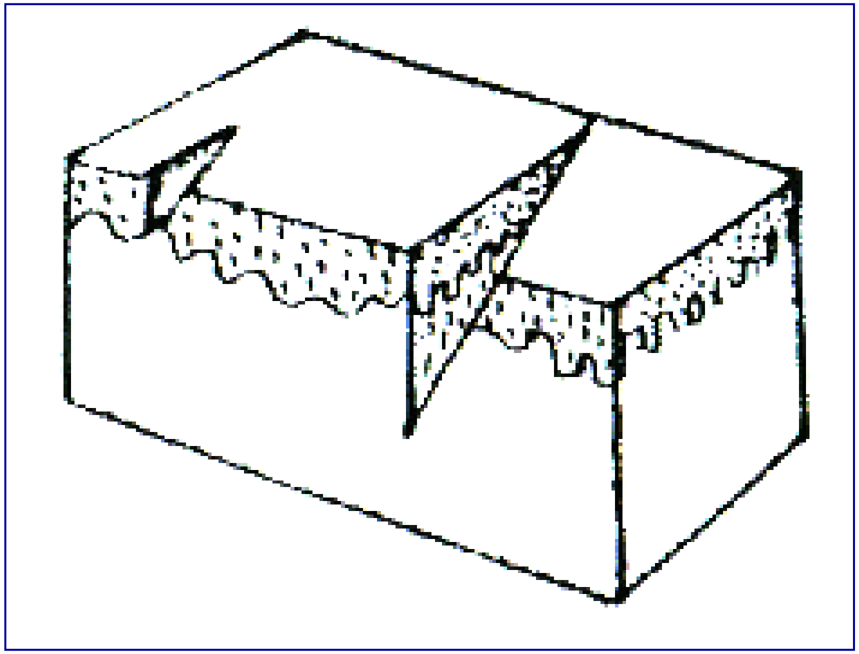
Secondary Lesion



FISSURE

a linear split in epidermis or dermis, with sharply defined **vertical** walls, over a **joint** or along a skin **crease** (skin isn´t flexible due to mycosis, eczema, psoriasis…)

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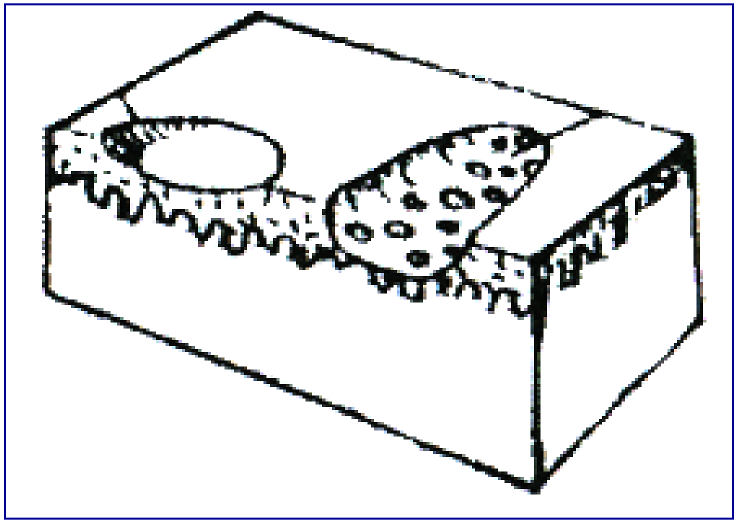
Secondary Lesions



EROSION

a partial loss of **epidermis**, heals without scarring

EXCORIATION

a localized damage to the skin due to **scratching**

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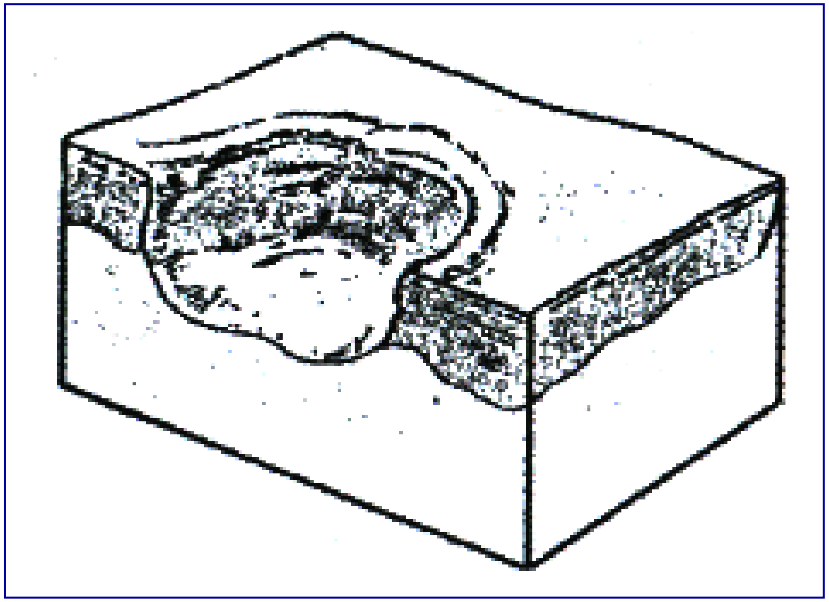
Secondary Lesion



ULCER

a full thickness loss
of **epidermis end dermis**,
which will heal with scarring

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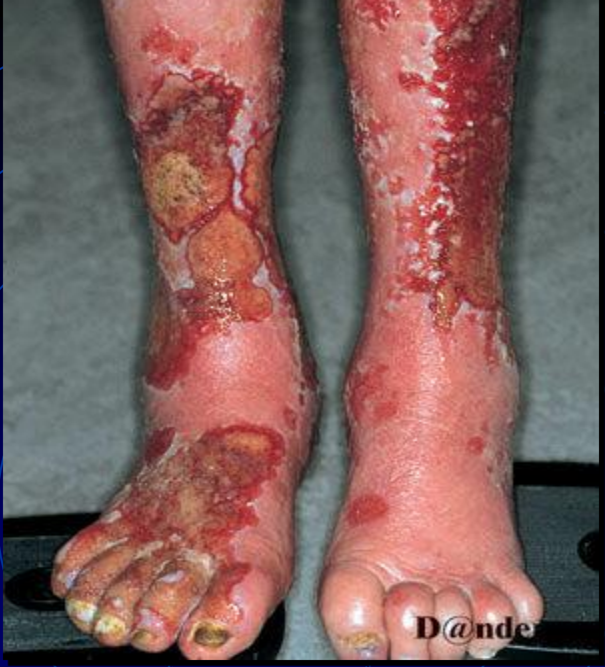
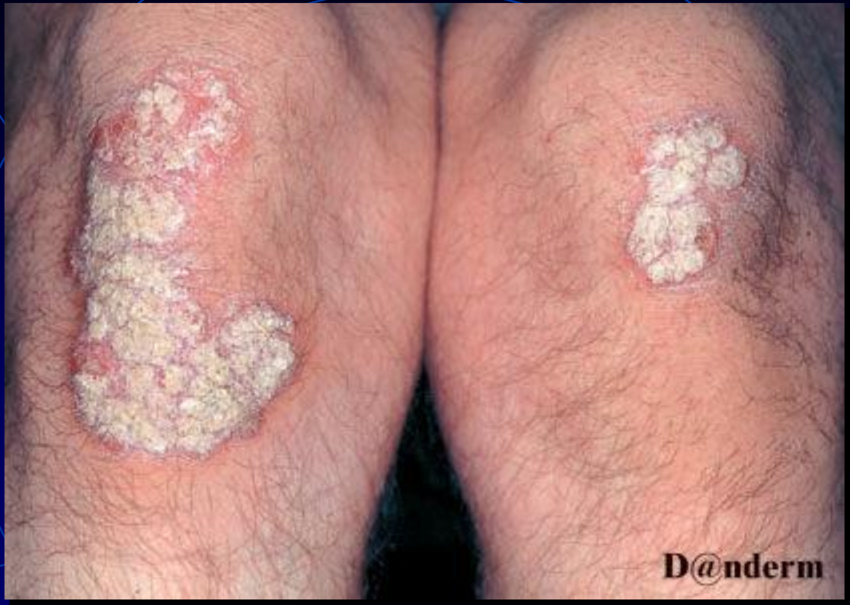
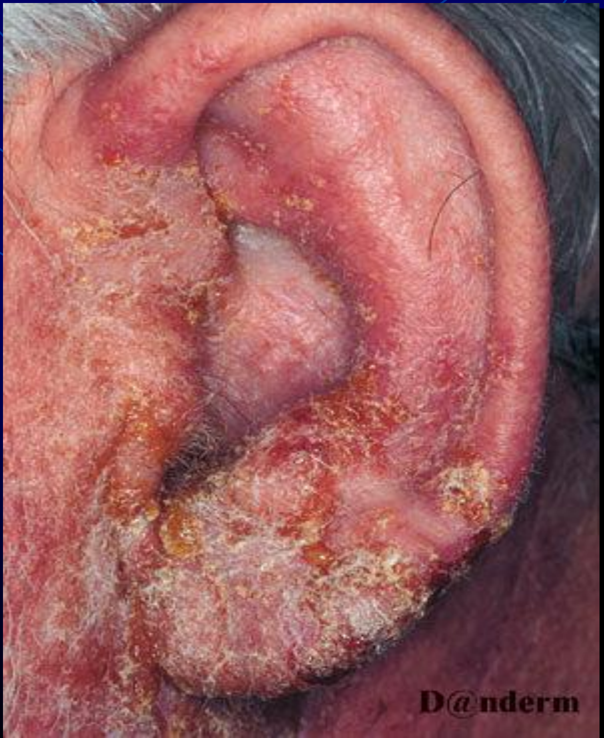

D@nde


D@nderm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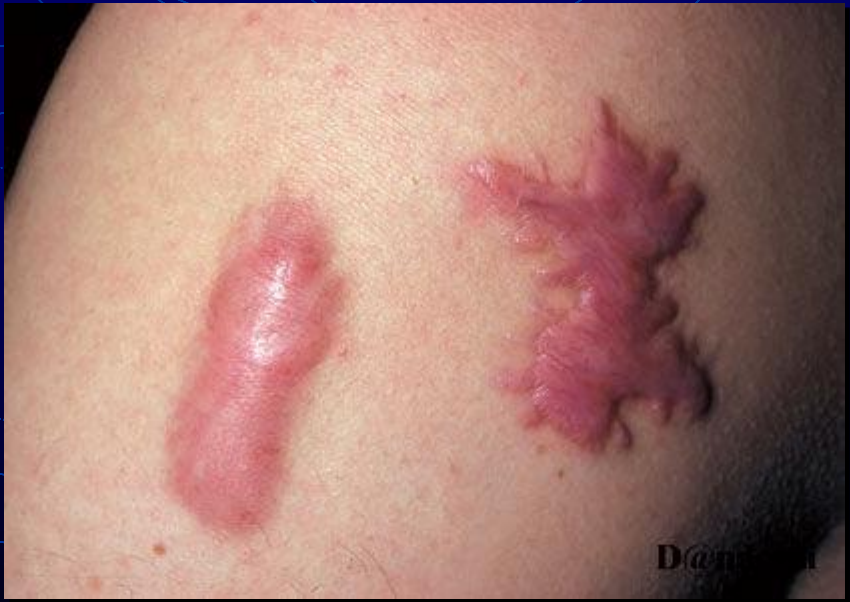
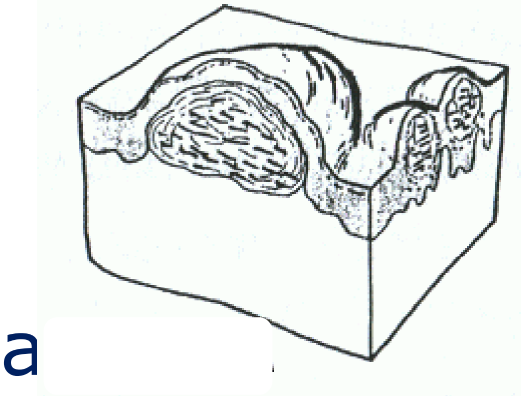
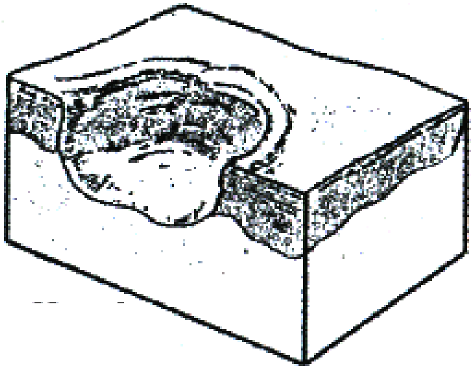




Blisters, pustules:

- **blister roof** – thin, thick, flaccid…
- **content** – clear, turbid, haemorrha

Ulcers:



- **base** – deep x shallow, uneven, clear, coated, with granulation tiss
- **edge** – undermined, collowed, flat adjacent, with epitelization…

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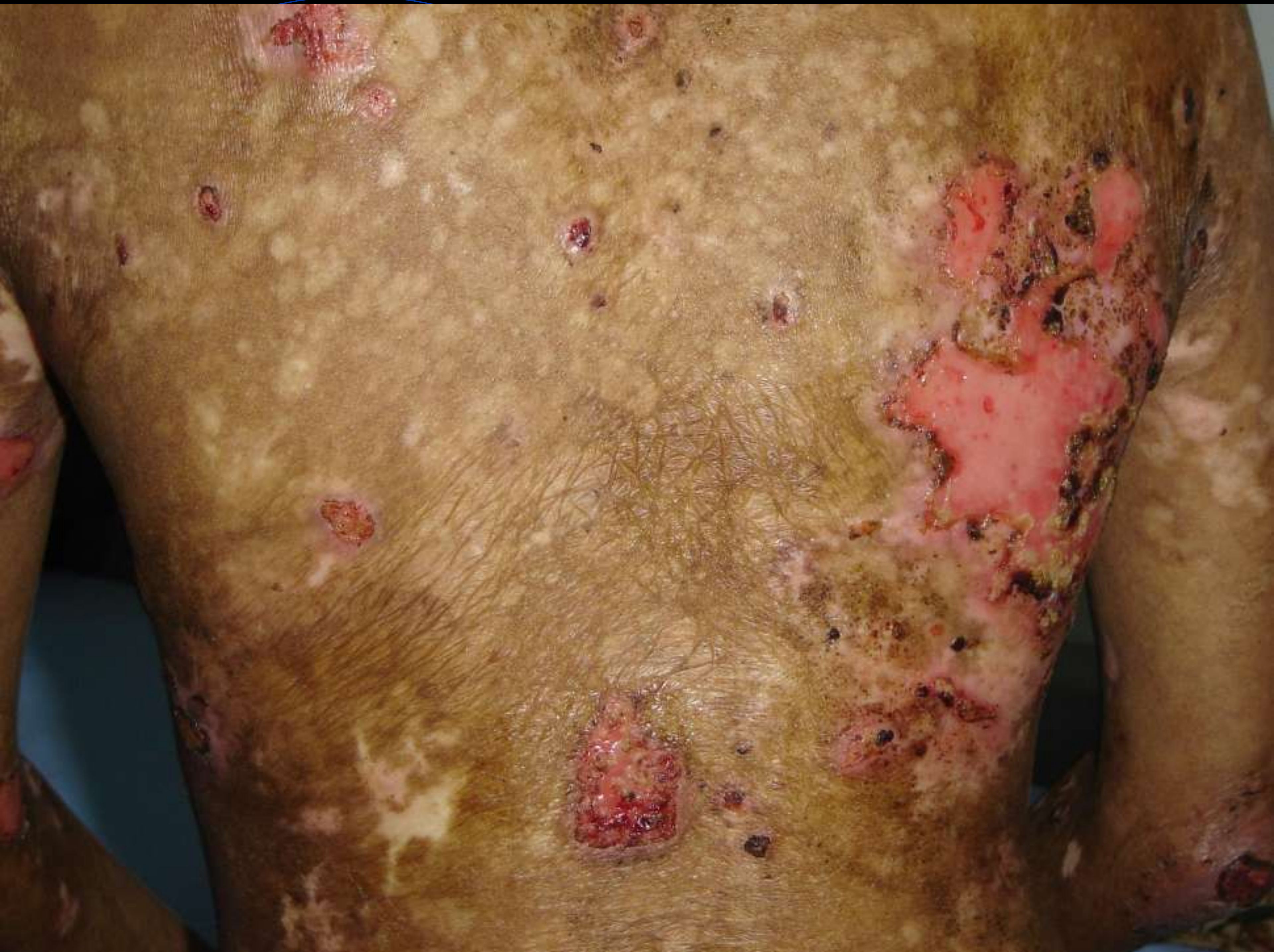

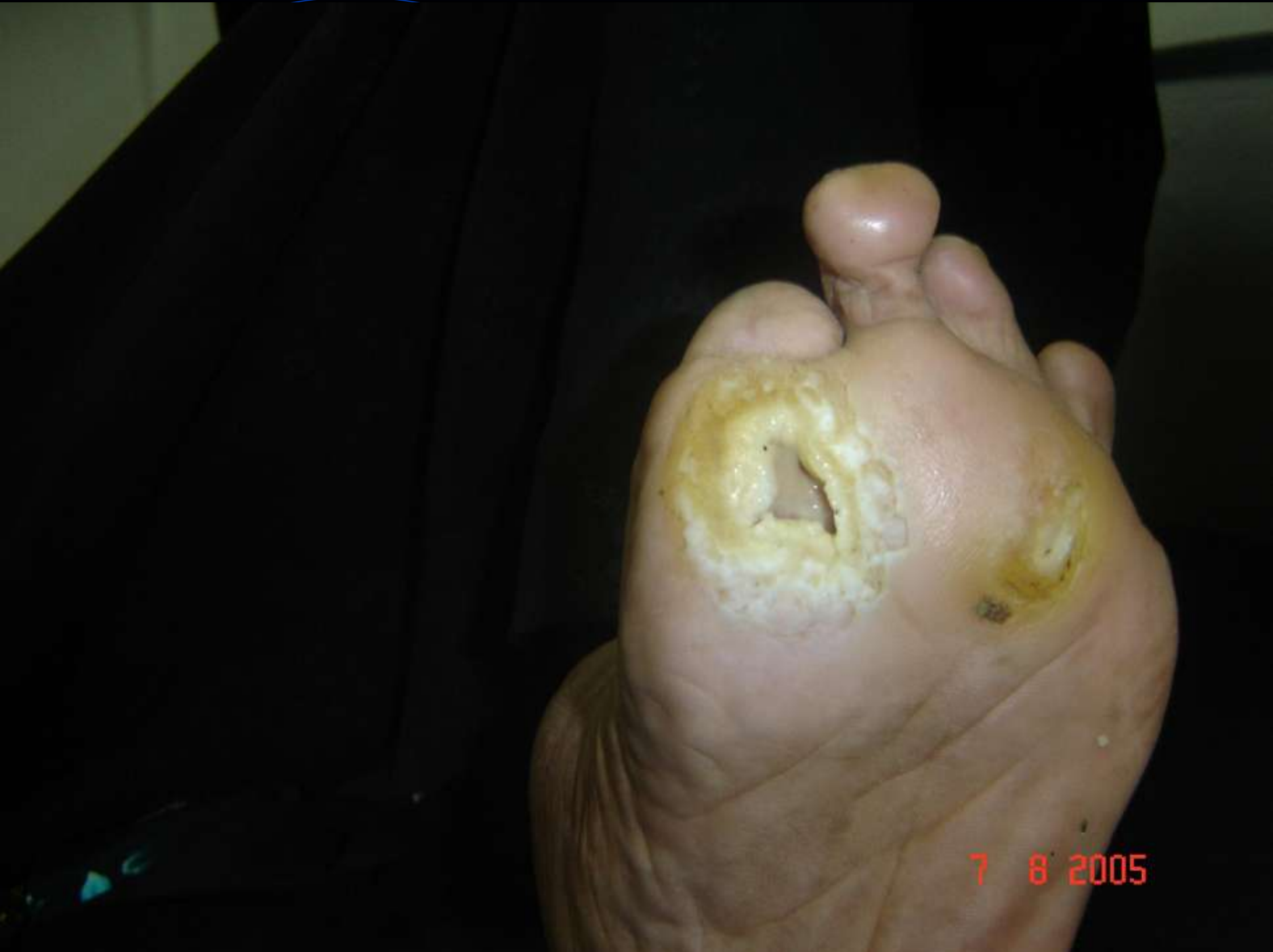

7 8 2005

Lesions can be:

- ✓ **single** or **multiple**
- ✓ **grouped** (multiple lesions grouped in one area… …are confluent in one area)
- ✓ **disseminated** (exanthema = widespread discrete lesions)
- ✓ **arranged** (lesions form some configuration – e.g. in

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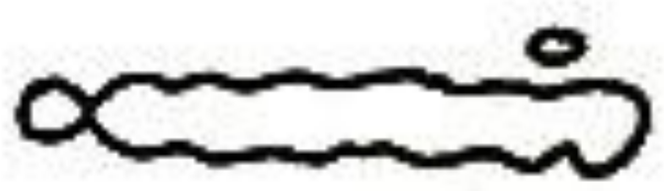
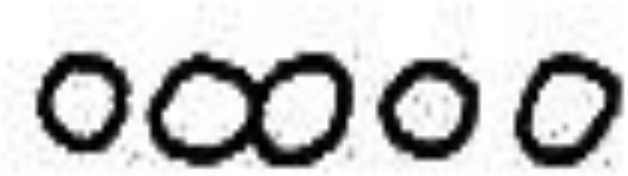
Arrangements of Lesions

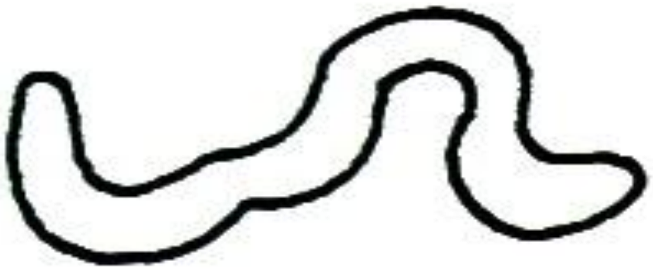
linear configuration





moniliform configuration

serpiginous configuration



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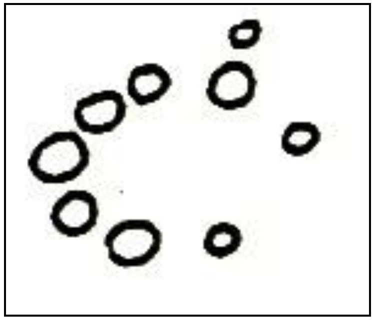
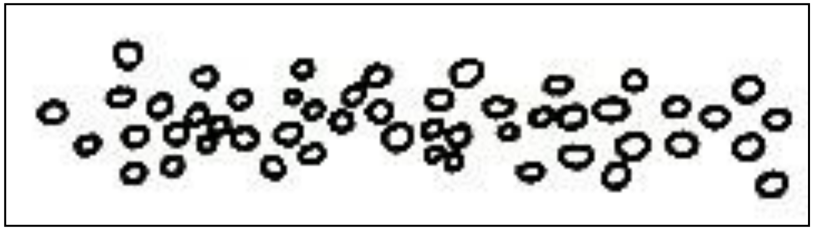
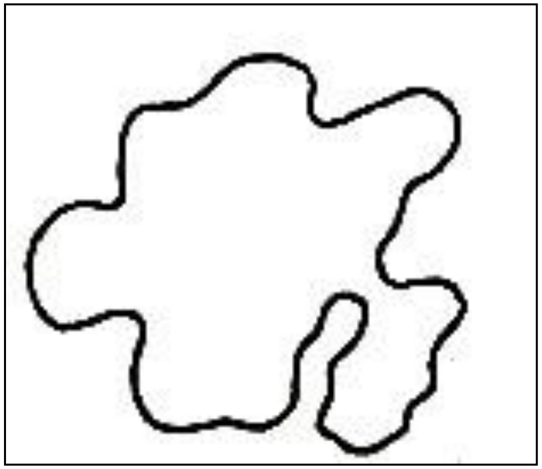
Arrangements of Lesions





zosteriform configuration

herpetiform conf.



geografical configuration

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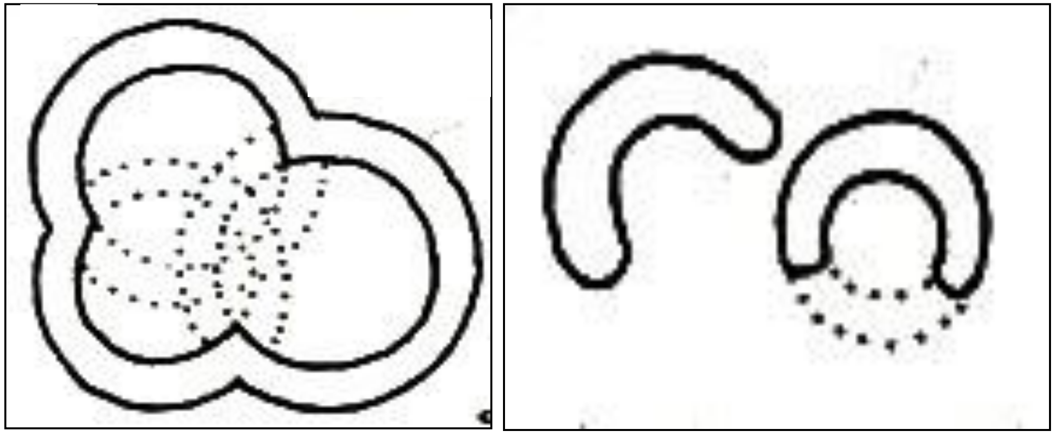
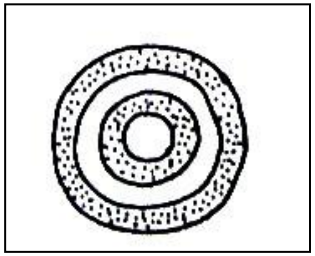
Arrangements of Lesions

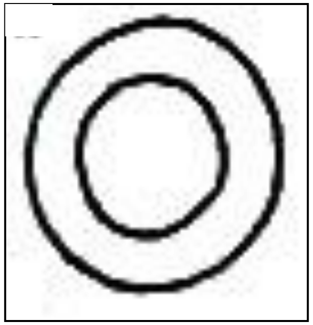


circinate, semicircinate configuration



iris form

(concentric circles)



annular conf.

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Other Changes

…erythroderma, erythema

atrophy, lichenification

papilomatosis, pachydermatosis

hyperpigmentation, depigmentation

scars….

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Describing of Lesions:

- **sites involved and distribution** (a-symmetrical, predilection sites; arrangement, single or multiple)
- **size** (mm, cm)
- **shape** (round, oval, irregular, pedunculated…)
- **surface features** (smooth, rough, shiny, dull, squamous, uneven…)
- **demarcation - border of lesion** (well defined or circumscribed, poorly defined…)
- **color** (pink, red, purple, white, brown, yellowish…)

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Disorders of the sweat glands

ASST. PROF. **AL-MURIESH MAHER**, MBBS, MSc, M.D

Conflict of interest Statement

I have no conflicts of interest to declare

Presentation plan

- Overview
- Structure and function
- Eccrine gland and its disorder
- Apocrine gland and its disorder
- Bromhidrosis
- Chromhidrosis

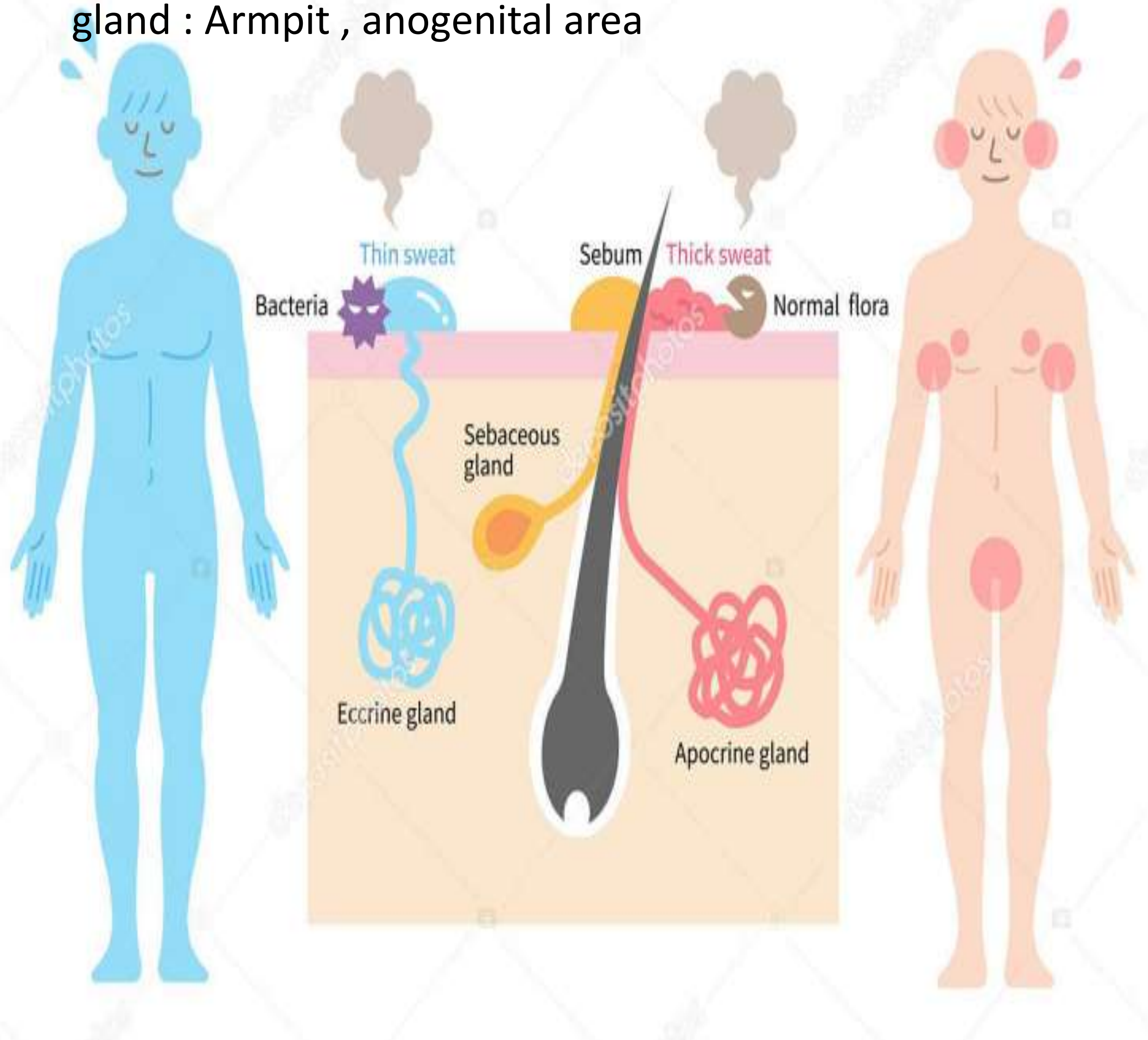
Overview

Eccrine sweat glands: are distributed over the whole skin surface **Except:**
the lips, external ear canal, clitoris,and labia minora.

Acetylcholine is the major stimulus of eccrine sweat glands secreted by sympathetic nerves

Apocrine sweat glands: in the adult are localized to the axillae, perianal region, and areolae of the breasts. Apocrine glands enlarge during puberty and their activity is **androgen** dependent. Adrenergic stimulation controls apocrine gland secretion.

Eccrine sweat glands : Everywhere (**almost**) Apocrine gland : Armpit , anogenital area


Thin sweat
Sebum
Thick sweat
Bacteria
Normal flora
Sebaceous gland
Eccrine gland
Apocrine gland

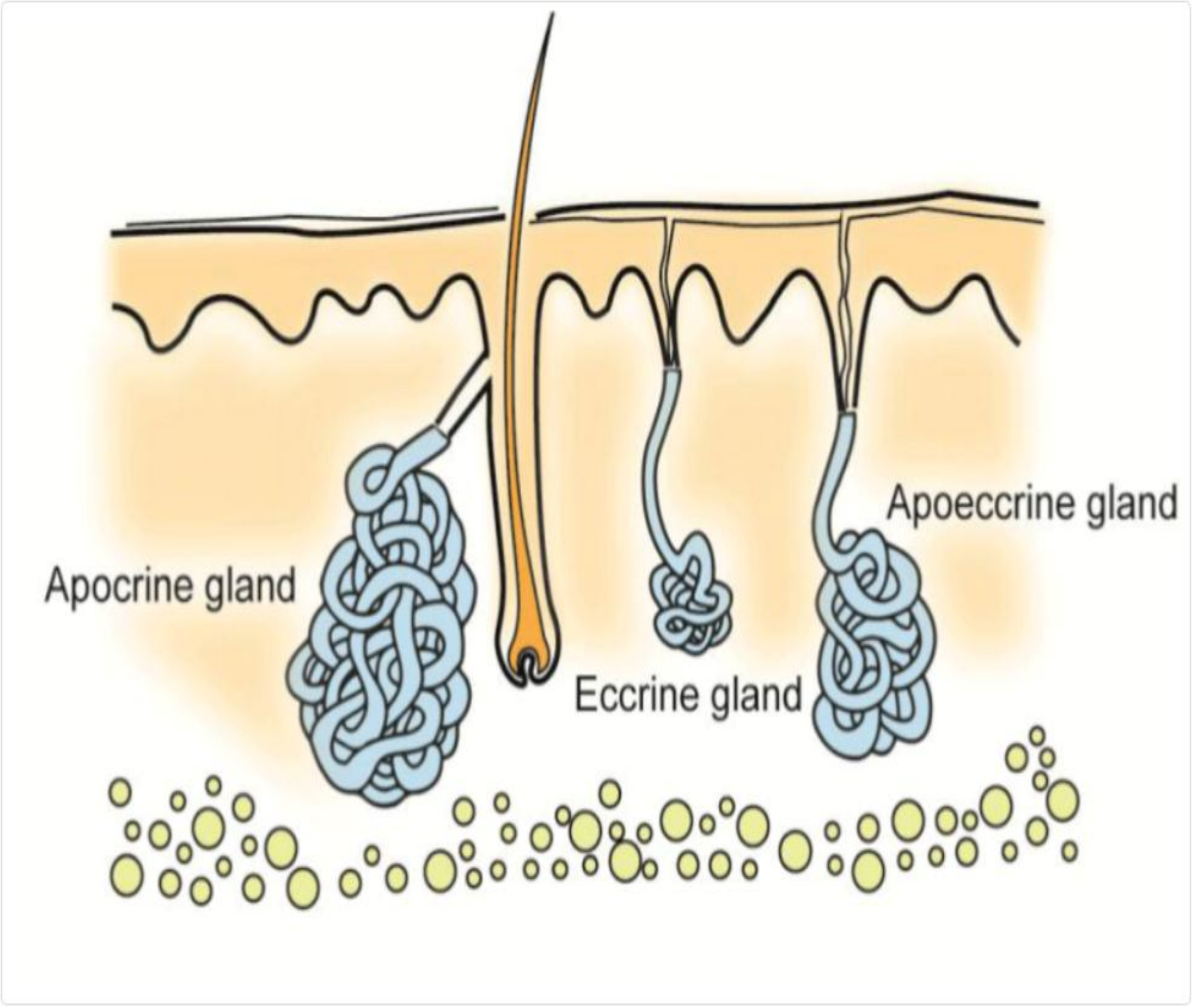
Is there a third sweat gland ?

Yes,

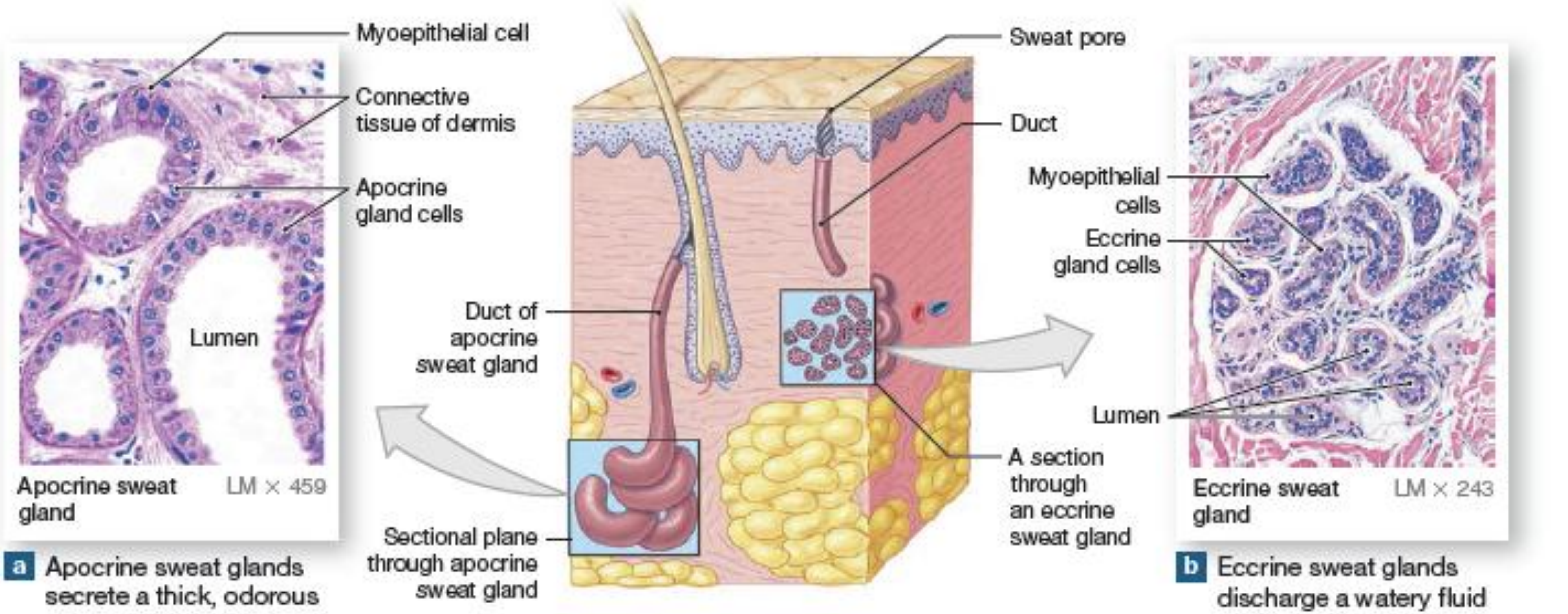
Apoeccrine gland share properties with both eccrine and apocrine glands.

Distribution: the axillary region.

connects to and empties sweat directly onto skin surface.


Apocrine gland
Eccrine gland
Apoeccrine gland

Structure and function


Myoepithelial cell
Connective tissue of dermis
Apocrine gland cells
Lumen
Apocrine sweat gland
LM × 459
Duct of apocrine sweat gland
Sectional plane through apocrine sweat gland
Sweat pore
Duct
A section through an eccrine sweat gland
Myoepithelial cells
Eccrine gland cells
Lumen
Eccrine sweat gland
LM × 243

a Apocrine sweat glands secrete a thick, odorous fluid into hair follicles.

b Eccrine sweat glands discharge a watery fluid onto the surface of the skin.

Disorders of eccrine sweat glands

Disorders of eccrine sweating can be subdivided

into two main categories:

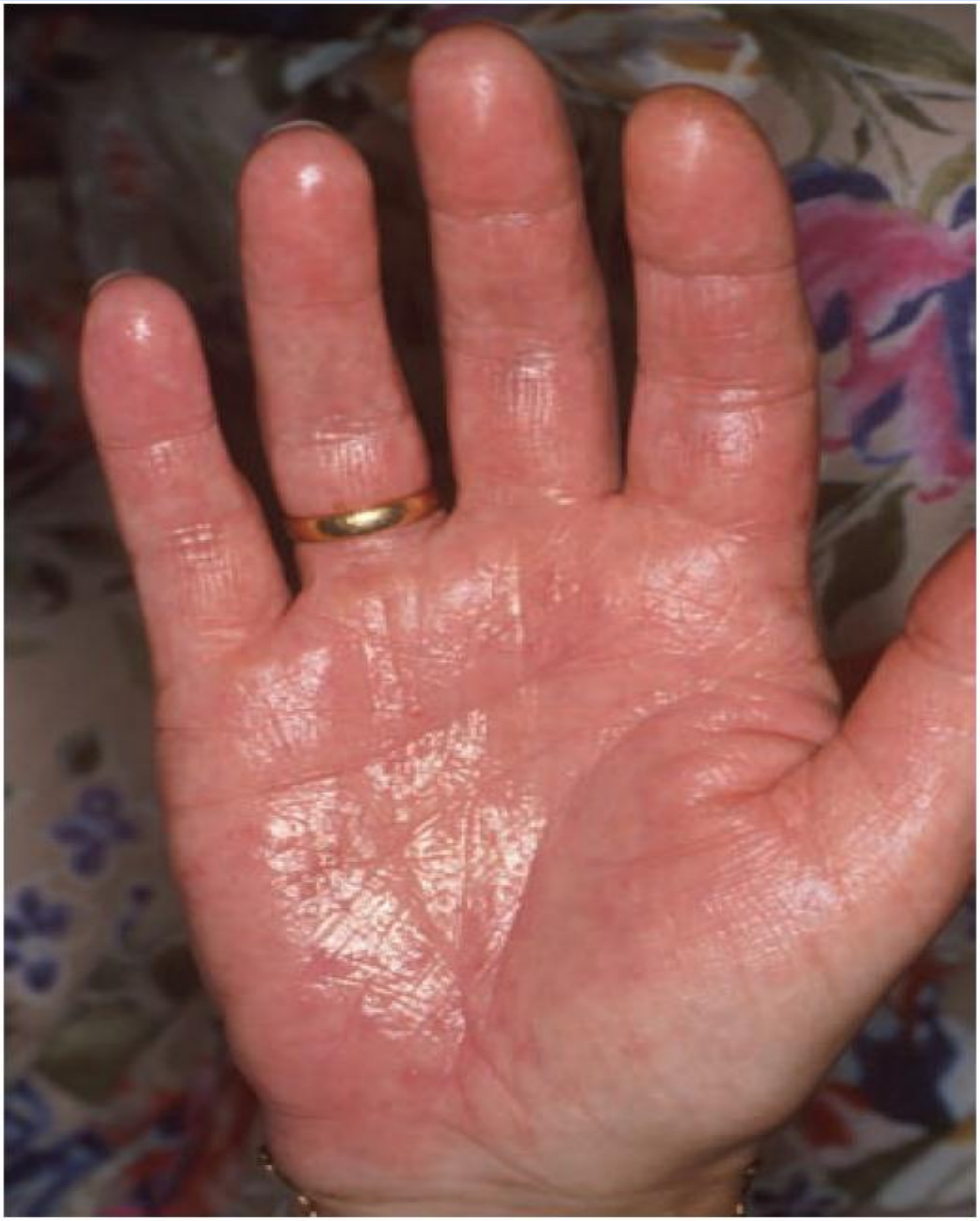
excessive sweating (hyperhidrosis)

reduced sweating (hypohidrosis or anhidrosis)

Hyperhidrosis

Hyperhidrosis is a condition of excessive sweating beyond what is physiologically necessary to maintain normal body temperature.

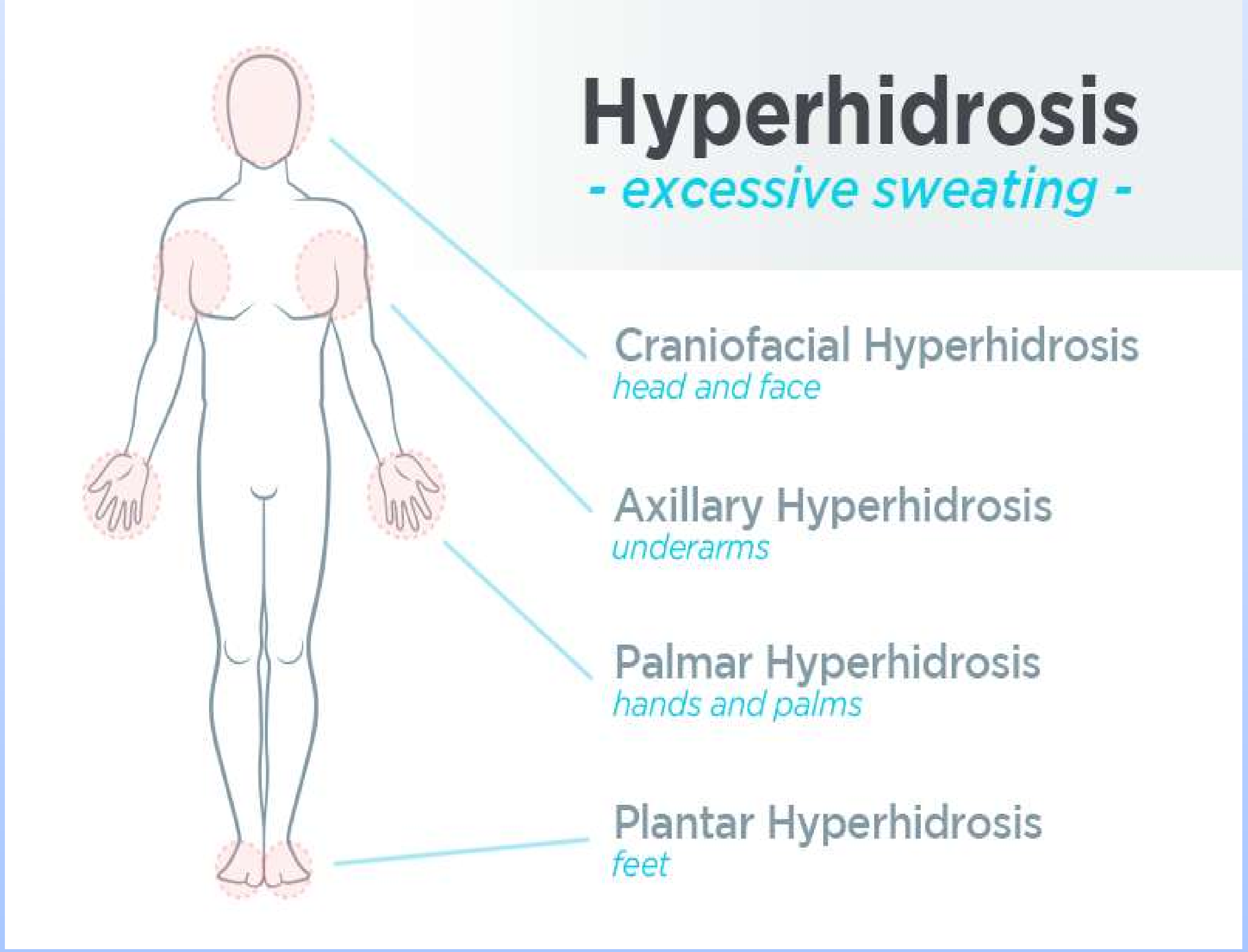
The prevalence at 4.8%, represents around 15.3 million people in the United States. Hyperhidrosis can be further subdivided into **primary** and **secondary** hyperhidrosis.



Hyperhidrosis

➢ **Primary hyperhidrosis** is a neurologic disorder that manifests as excessive sweating at baseline in various anatomic locations.

- It affects males and females equally,
- begins in childhood (palmar plantar) or during puberty (axillary).


Hyperhidrosis
- excessive sweating -
Craniofacial Hyperhidrosis
head and face
Axillary Hyperhidrosis
underarms
Palmar Hyperhidrosis
hands and palms
Plantar Hyperhidrosis
feet

Hyperhidrosis

- **Secondary hyperhidrosis** is caused by or associated with systemic disorder.

- Localized or generalized.
- Multiple causes
- Can be divided into categories based on the source of the neural impulse driving the response: **c**ortical, **h**ypothalamic, **m**edullary, **s**pinal cord, or **l**ocal.
- Medications may also be associated with hyperhidrosis

Hyperhidrosis

- Disorders associated with cortical (emotional) hyperhidrosis
- Hypothalamic (thermoregulatory) hyperhidrosis
- Medullary (gustatory) hyperhidrosis
- Autonomic dysreflexia

Hyperhidrosis

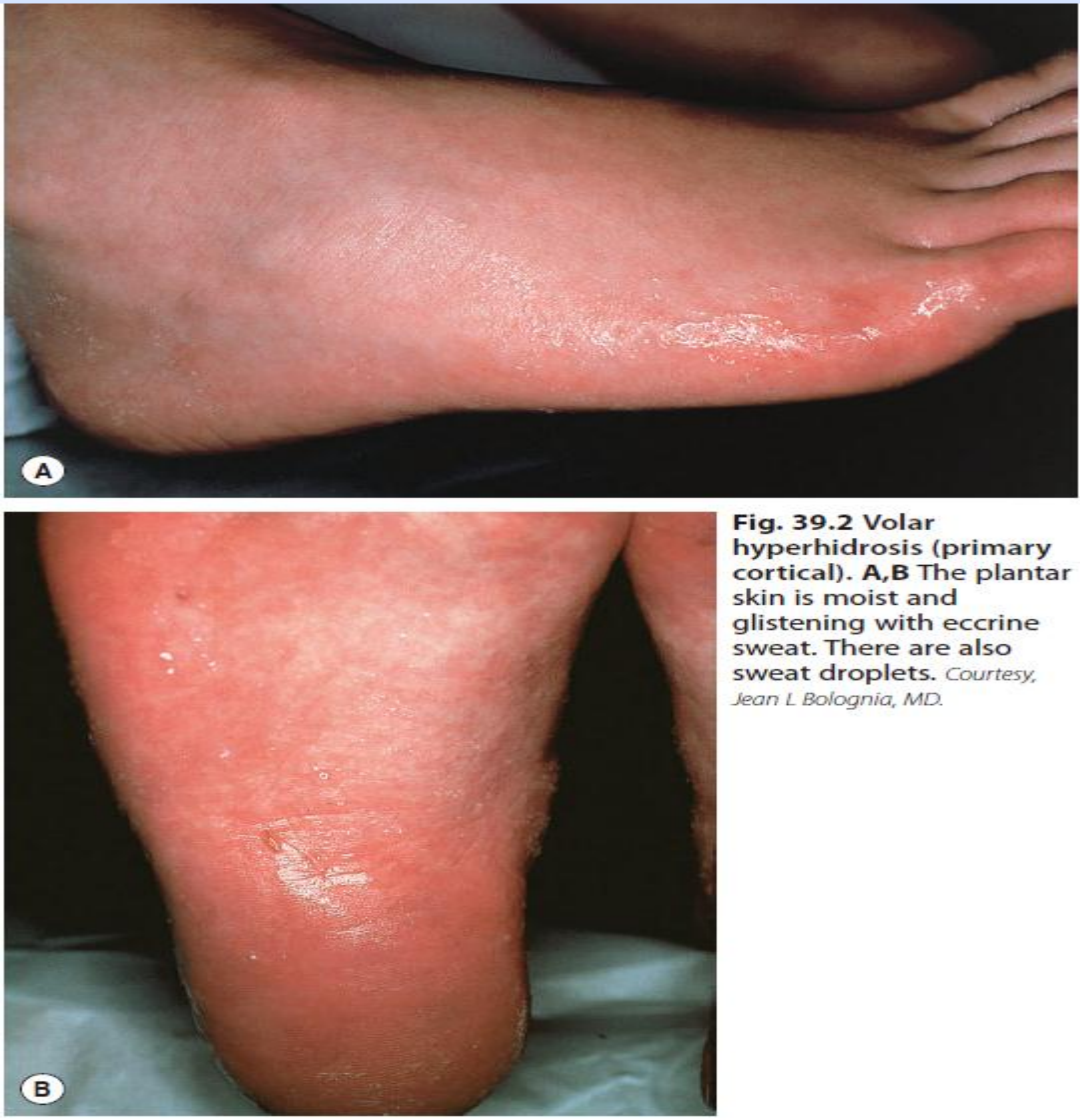


Fig. 39.2 Volar hyperhidrosis (primary cortical). **A,B** The plantar skin is moist and glistening with eccrine sweat. There are also sweat droplets. *Courtesy, Jean L Bolognia, MD.*

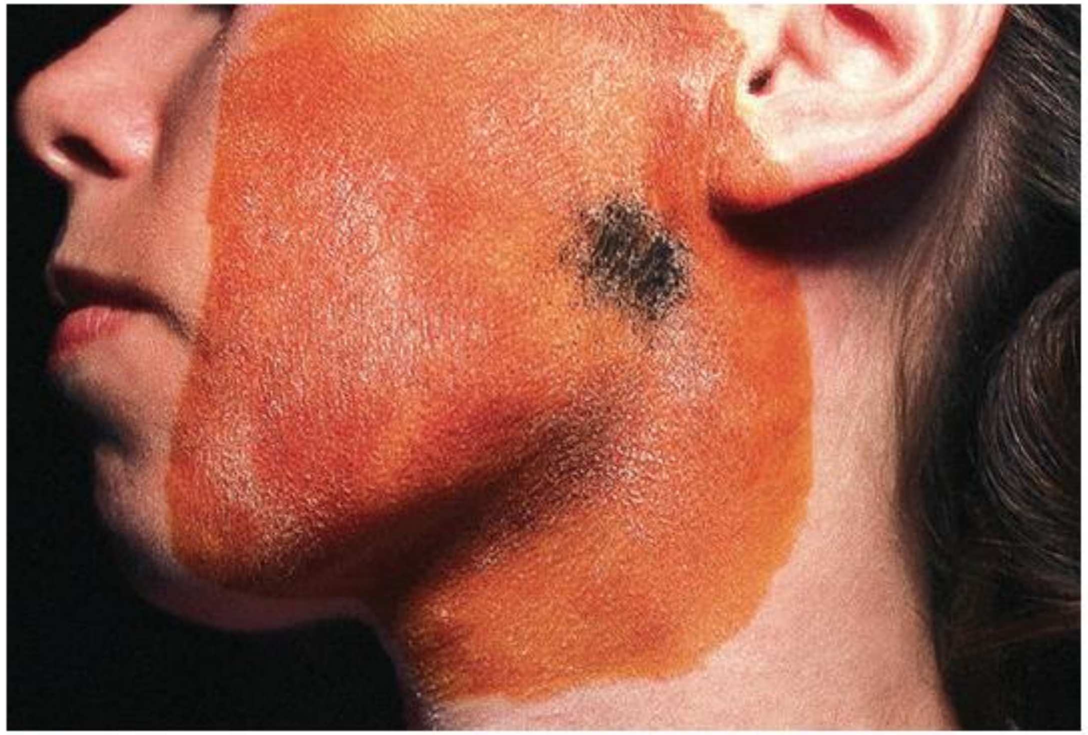
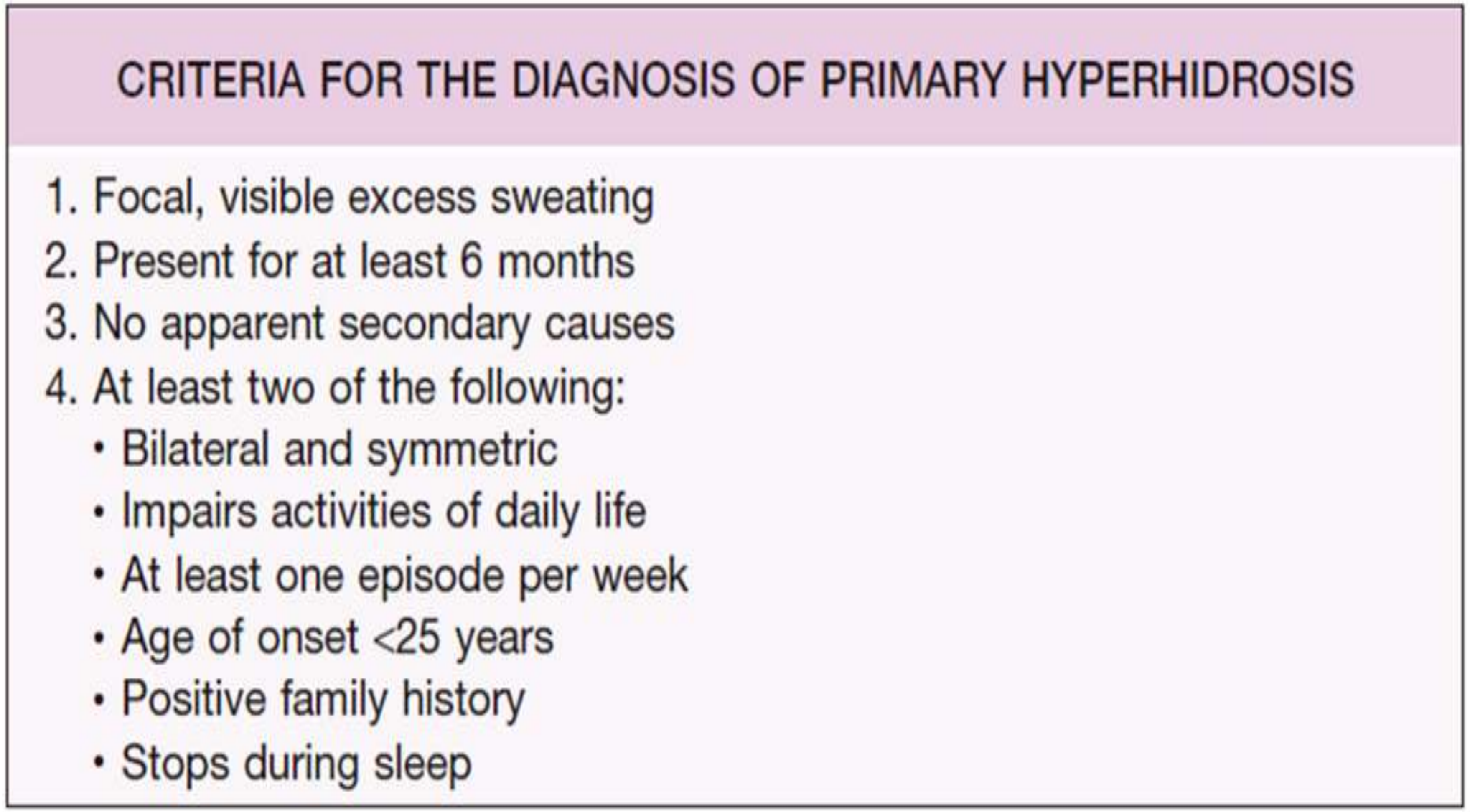


Fig. 39.3 Gustatory sweating in the auriculotemporal (Frey) syndrome, as a consequence of parotid surgery. The blue–black area represents sweating (starch–iodine technique). Salivary stimulation induced this sweating response.

Diagnosis

- The first step is to differentiate between primary and secondary hyperhidrosis.
  - Medical history
  - Laboratory testing
  - Hyperhidrosis can be documented by colorimetric and gravimetric methods

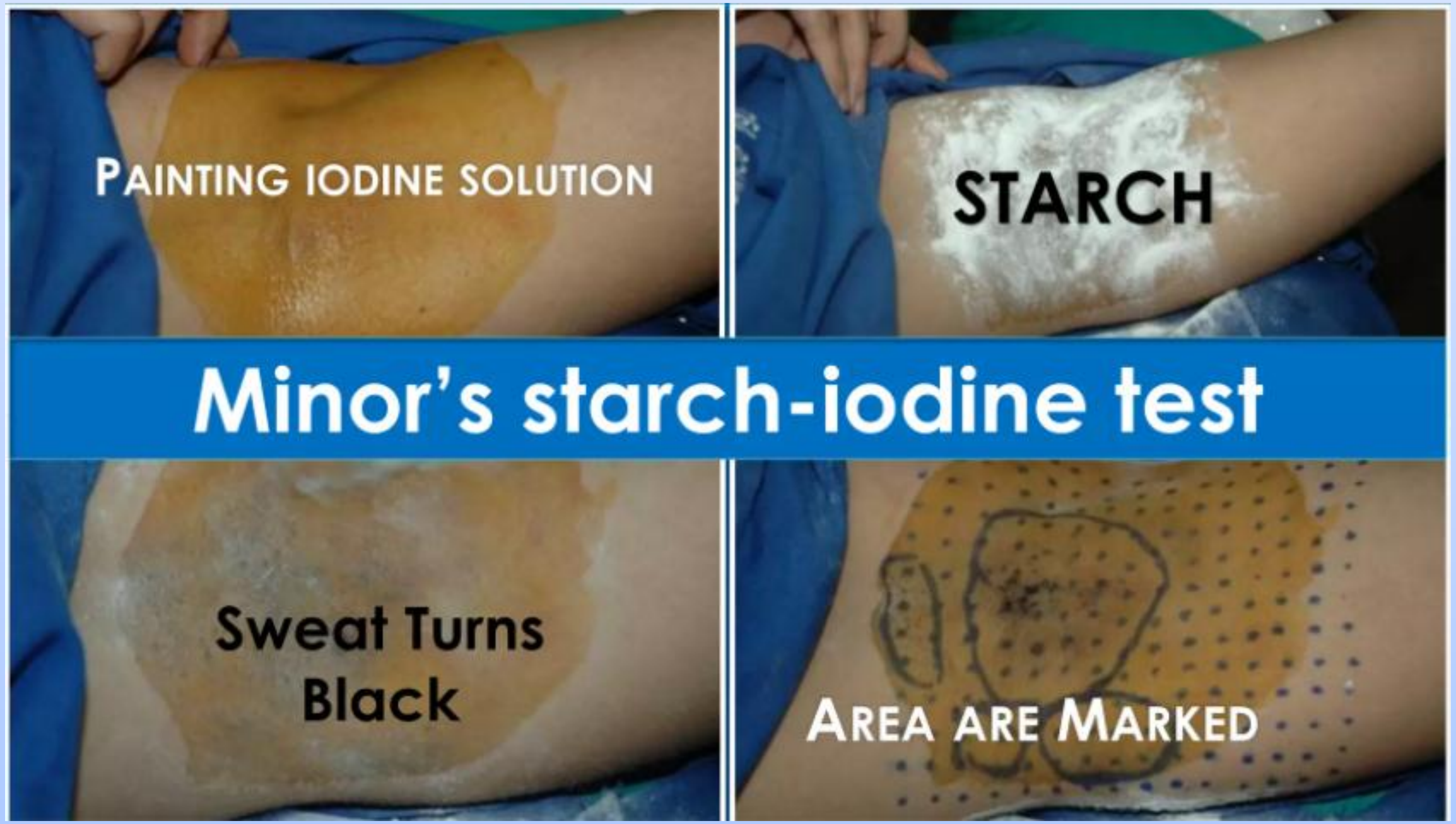


CRITERIA FOR THE DIAGNOSIS OF PRIMARY HYPERHIDROSIS

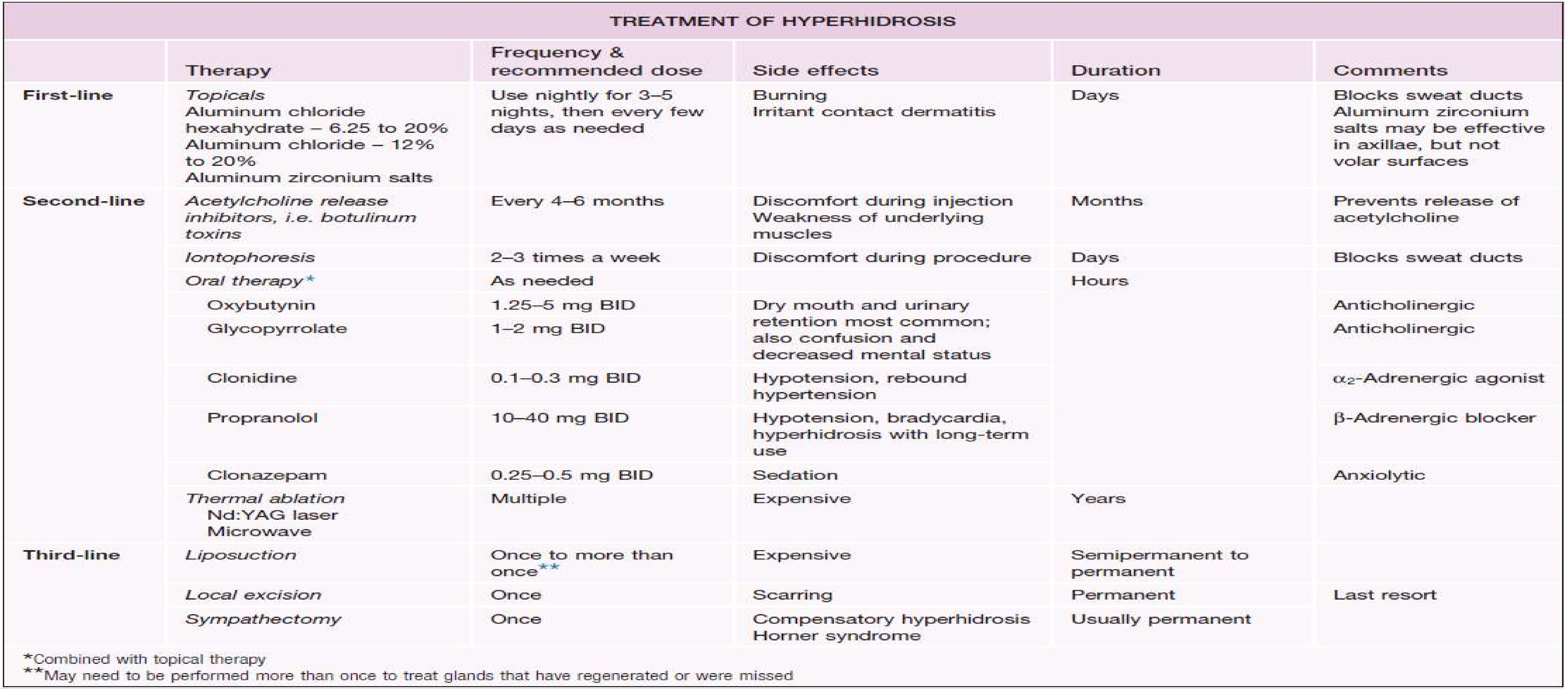
1. Focal, visible excess sweating
2. Present for at least 6 months
3. No apparent secondary causes
4. At least two of the following:
   - Bilateral and symmetric
   - Impairs activities of daily life
   - At least one episode per week
   - Age of onset <25 years
   - Positive family history
   - Stops during sleep

Table 39.1 Criteria for the diagnosis of primary hyperhidrosis. *Adapted from Hornberger J, Grimees K, Naumann M, et al. Recognition, diagnosis and treatment of primary focal hyperhidrosis. J Am Acad Dermatol. 2004;51:274–86.*

Diagnosis


Painting iodine solution
Starch
Minor's starch-iodine test
Sweat Turns Black
Area are Marked

Treatment



TREATMENT OF HYPERHIDROSIS

	Therapy	Frequency & recommended dose	Side effects	Duration	Comments
First-line	*Topicals* Aluminum chloride hexahydrate – 6.25 to 20% Aluminum chloride – 12% to 20% Aluminum zirconium salts	Use nightly for 3–5 nights, then every few days as needed	Burning Irritant contact dermatitis	Days	Blocks sweat ducts Aluminum zirconium salts may be effective in axillae, but not volar surfaces
Second-line	*Acetylcholine release inhibitors, i.e. botulinum toxins*	Every 4–6 months	Discomfort during injection Weakness of underlying muscles	Months	Prevents release of acetylcholine
	Iontophoresis	2–3 times a week	Discomfort during procedure	Days	Blocks sweat ducts
	Oral therapy*	As needed		Hours	
	Oxybutynin	1.25–5 mg BID	Dry mouth and urinary retention most common; also confusion and decreased mental status		Anticholinergic
	Glycopyrrolate	1–2 mg BID			Anticholinergic
	Clonidine	0.1–0.3 mg BID	Hypotension, rebound hypertension		α_2-Adrenergic agonist
	Propranolol	10–40 mg BID	Hypotension, bradycardia, hyperhidrosis with long-term use		β-Adrenergic blocker
	Clonazepam	0.25–0.5 mg BID	Sedation		Anxiolytic
	Thermal ablation Nd:YAG laser Microwave	Multiple	Expensive	Years	
Third-line	*Liposuction*	Once to more than once**	Expensive	Semipermanent to permanent	
	Local excision	Once	Scarring	Permanent	Last resort
	Sympathectomy	Once	Compensatory hyperhidrosis Horner syndrome	Usually permanent	

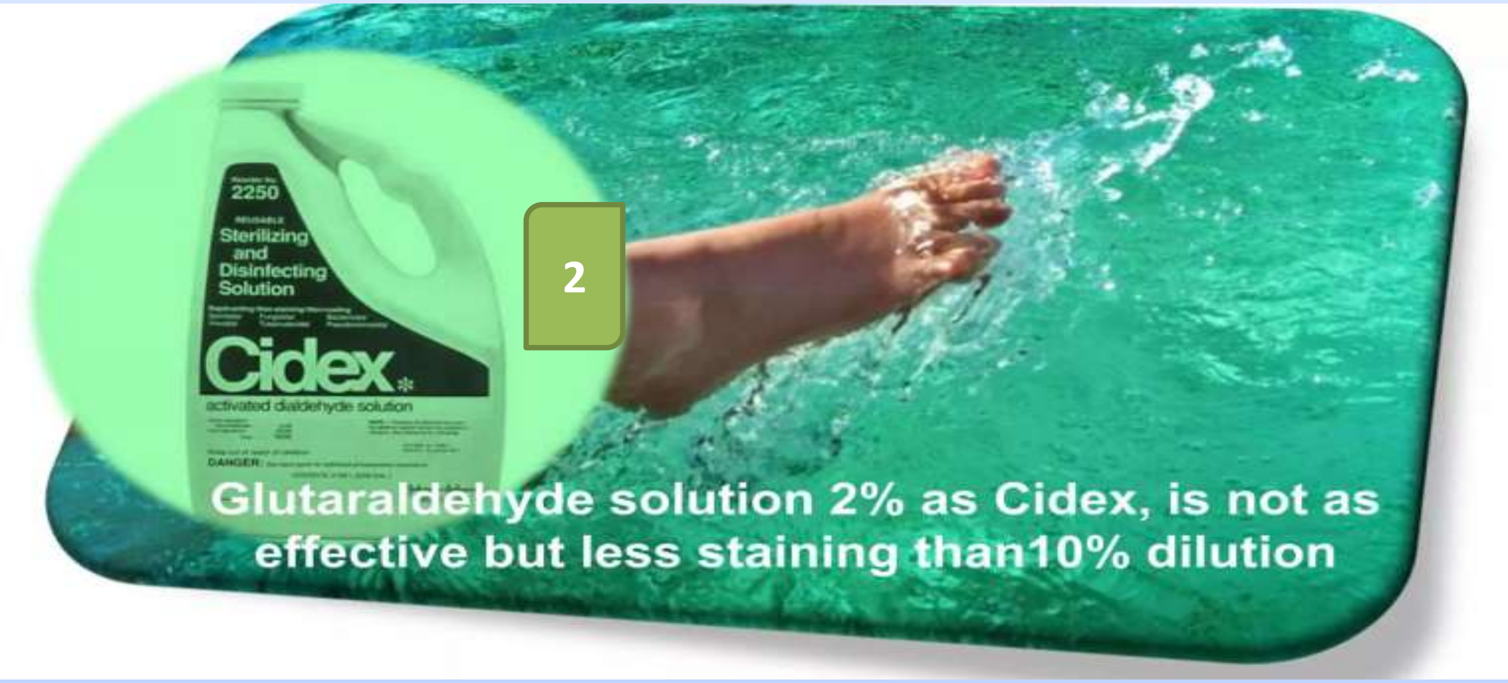
*Combined with topical therapy

**May need to be performed more than once to treat glands that have regenerated or were missed

1

Topical treatment

Prescription and specialty antiperspirants often contain aluminum chloride hexahydrate


2
2250
Sterilizing and Disinfecting Solution
Cidex
activated dialdehyde solution
DANGER

Glutaraldehyde solution 2% as Cidex, is not as effective but less staining than10% dilution

3
BOTOX
VISTABEL
Allergan-Einheiten
ALLERGAN
Botulinum Toxin Type A
BOTOX
Purified Neurotoxin Complex
100 UNITS
BOTOX Cosmetic

Treating excessive sweating of the armpits, hands, feet, and face is safe and effective

4

Iontophoresis

Water iontophoresis is the method of choice for the initial treatment of palmar and plantar hyperhidrosis

4

Iontophoresis

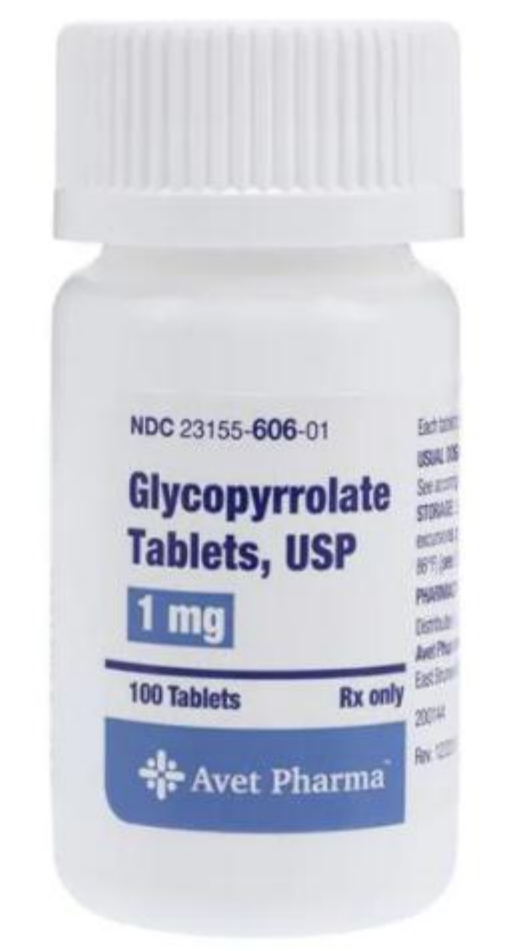
Treatment involves putting the affected areas (usually hands and/or feet) into a small container filled with tap water with backing soda or anticholinergic

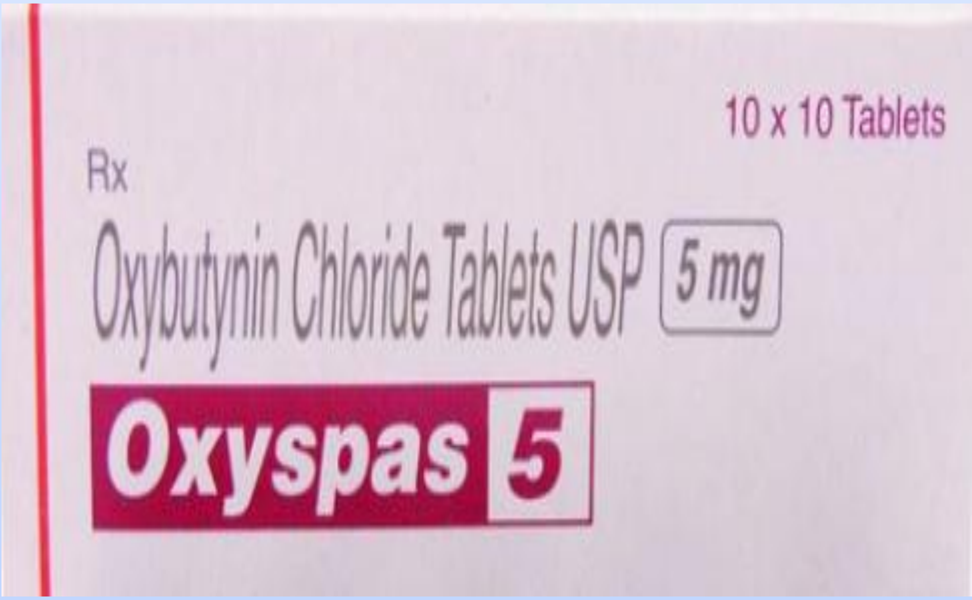
Systemic Treatment

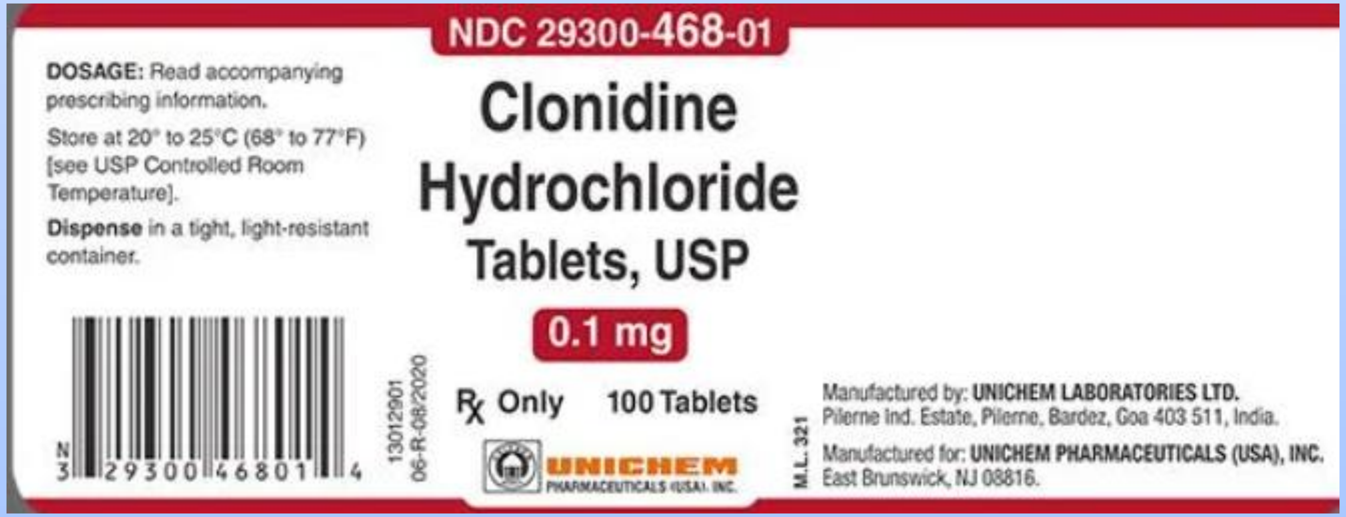
Take a pill
and Stop
Sweating is
not for
every case

2

Systemic treatment


NDC 23155-606-01
Glycopyrrolate Tablets, USP
1 mg
100 Tablets
Rx only
Avet Pharma


10 x 10 Tablets
Rx
Oxybutynin Chloride Tablets USP
5 mg
Oxyspas 5


NDC 29300-468-01
DOSAGE: Read accompanying prescribing information.
Store at 20° to 25°C (68° to 77°F) [see USP Controlled Room Temperature].
Dispense in a tight, light-resistant container.
Clonidine Hydrochloride Tablets, USP
0.1 mg
℞ Only
100 Tablets
UNICHEM PHARMACEUTICALS (USA), INC.
Manufactured by: UNICHEM LABORATORIES LTD. Pilerne Ind. Estate, Pilerne, Bardez, Goa 403 511, India.
Manufactured for: UNICHEM PHARMACEUTICALS (USA), INC. East Brunswick, NJ 08816.
M.L. 321
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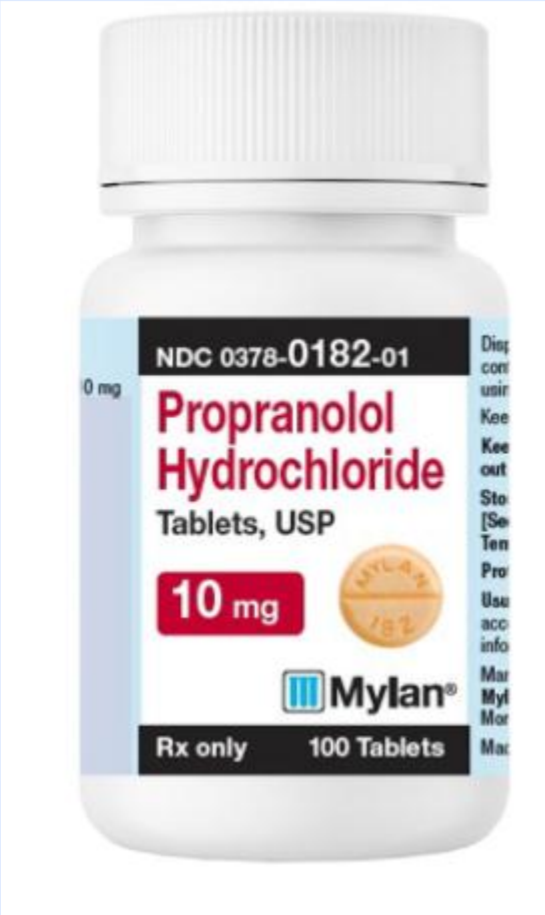

NDC 0378-0182-01
Propranolol Hydrochloride Tablets, USP
10 mg
MYLAN 182
Mylan®
Rx only
100 Tablets

3

Thermal ablation

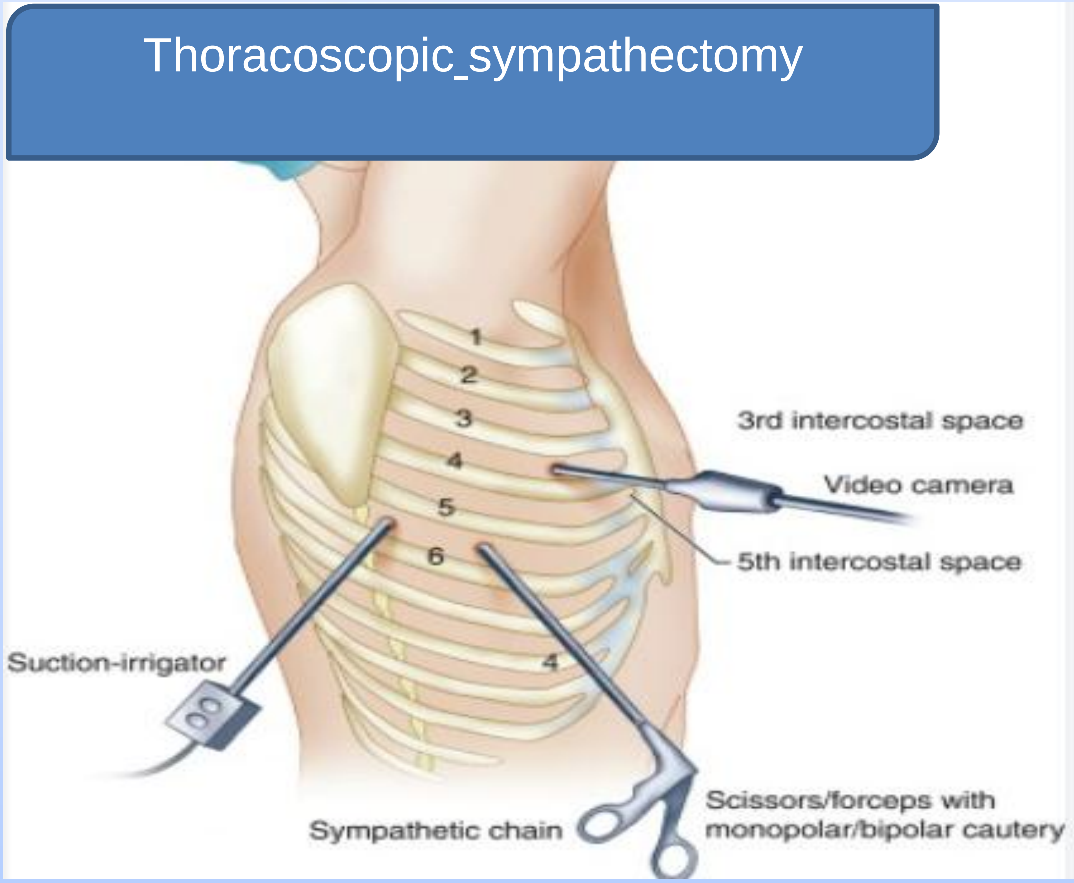
1 Nd:yag laser

2 microwave

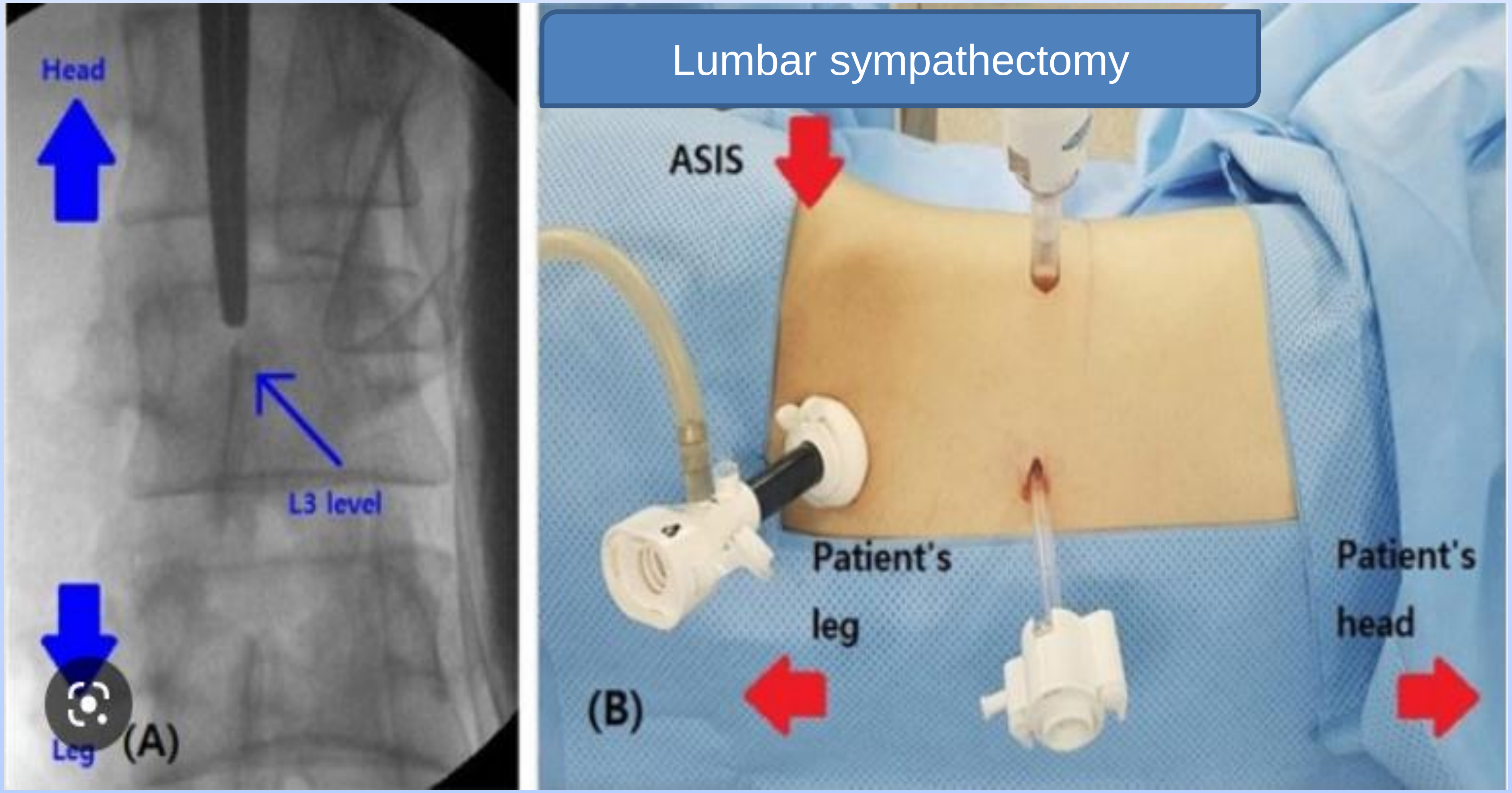
4

Surgical treatment

Thoracoscopic_sympathectomy


1
2
3
4
5
6
4
3rd intercostal space
Video camera
5th intercostal space
Suction-irrigator
Sympathetic chain
Scissors/forceps with monopolar/bipolar cautery

Lumbar sympathectomy


Head
L3 level
Leg
(A)
ASIS
Patient's leg
Patient's head
(B)

bland soap substitute

General tips and life style modification

- Bland soap substitute : moisturizing soap
- Avoid triggers
- Wearing loose clothes under the armpits
- Avoid clothes that more easily show up sweat marks
- Change your 100% cotton socks at least twice a day
- Use an absorbent foot powder twice a day
- Buy shoes that made of leather canvas or mesh rather than synthetic material
- Deodorant is not anti-perspirant

Change your 100% cotton socks at least twice a day

Use a mild, fragrant-free, antibacterial soap that can clean the skin without aggravating sweat.

Antiperspirants reduce the release of sweat, while deodorants mask unpleasant smells

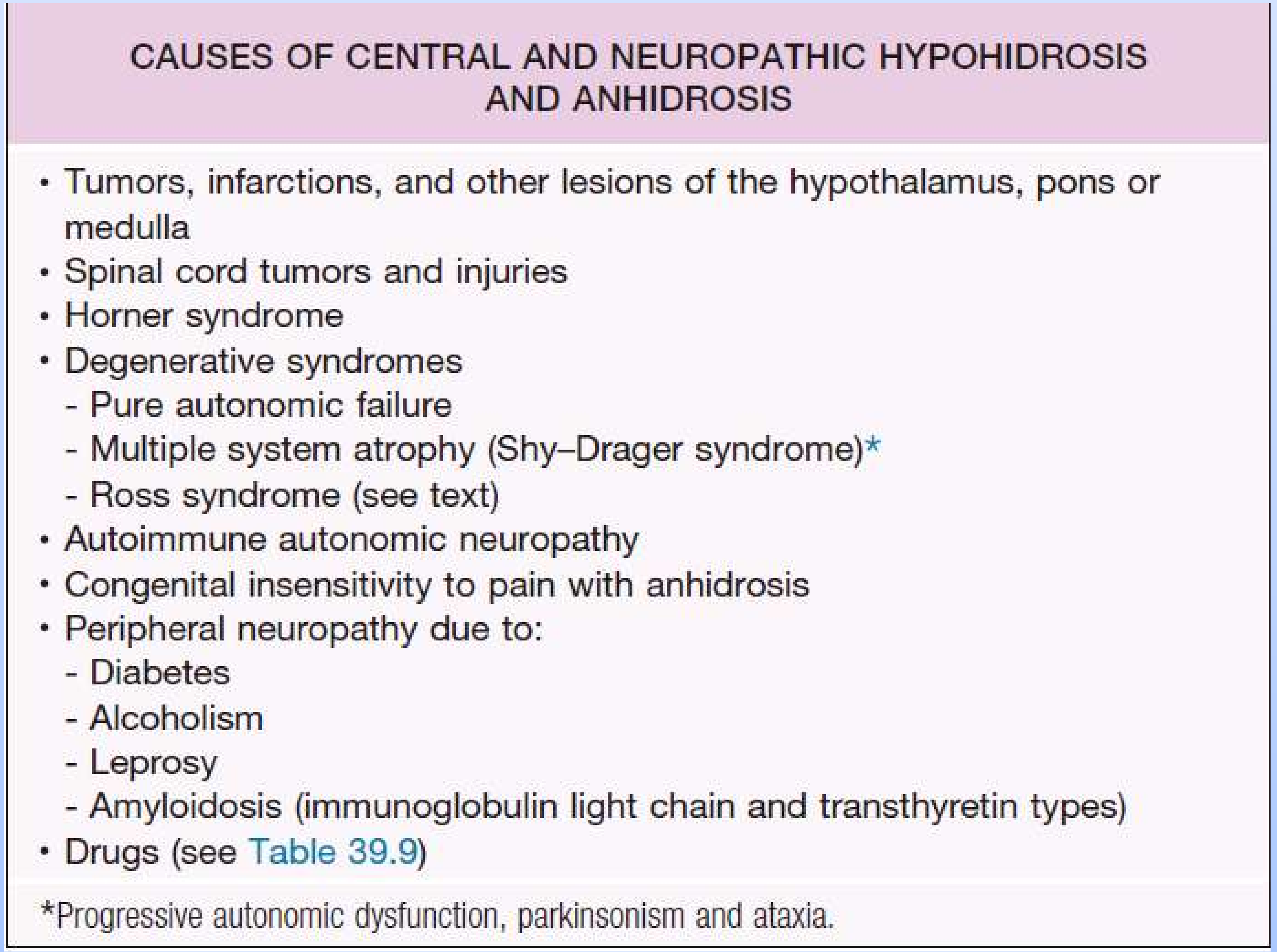
USE AN ABSORBENT FOOT POWDER TWICE DAILY

If possible, avoid triggers, which can make things worse such as heat or spicy food

Hypohidrosis and Anhidrosis

Hypohidrosis is the reduction in sweating. **Anihidrosis** is the absence of sweating

- Causes of hypo- and anhidrosis include central or neuropathic disruption of neural impulses
- Sweat gland abnormalities
- Medications
- Idiopathic



CAUSES OF CENTRAL AND NEUROPATHIC HYPOHIDROSIS AND ANHIDROSIS

- Tumors, infarctions, and other lesions of the hypothalamus, pons or medulla
- Spinal cord tumors and injuries
- Horner syndrome
- Degenerative syndromes
  - Pure autonomic failure
  - Multiple system atrophy (Shy–Drager syndrome)*
  - Ross syndrome (see text)
- Autoimmune autonomic neuropathy
- Congenital insensitivity to pain with anhidrosis
- Peripheral neuropathy due to:
  - Diabetes
  - Alcoholism
  - Leprosy
  - Amyloidosis (immunoglobulin light chain and transthyretin types)
- Drugs (see Table 39.9)

*Progressive autonomic dysfunction, parkinsonism and ataxia.

Diagnosis

Medical history

Examination of the patient’s skin may not reveal the presence or extent of anhidrosis.

Hot environment or exercise may be required, (excessive overheating must be avoided)

Colorimetric or gravimetric testing

Local intradermal injection of cholinergic drugs to stimulate sweating in small areas can also be utilized, but the risk of side effects

Testing for axon reflex sweating

Treatment

- The primary cause should be identified first, and any possible contributing medications should be discontinued.
- Keeping the patient in cool temperatures is crucial to avoid life-threatening overheating.
- It can also be helpful to wear wet clothing during physical activity; however, the effectiveness of external cooling may be compromised in patients with ectodermal dysplasia, presumably because of poor capillary dilation

Miliaria

Introduction

Miliaria encompasses a family of eccrine disorders all of which feature obstruction of the eccrine sweat duct.

لا يمكن عرض هذه الصورة حاليًا.

Etiology

- Occlusion and or inflammation of the eccrine sweat gland
- Hot and humid environment, strenuous physical exam , febrile illness , occlusion of the skin

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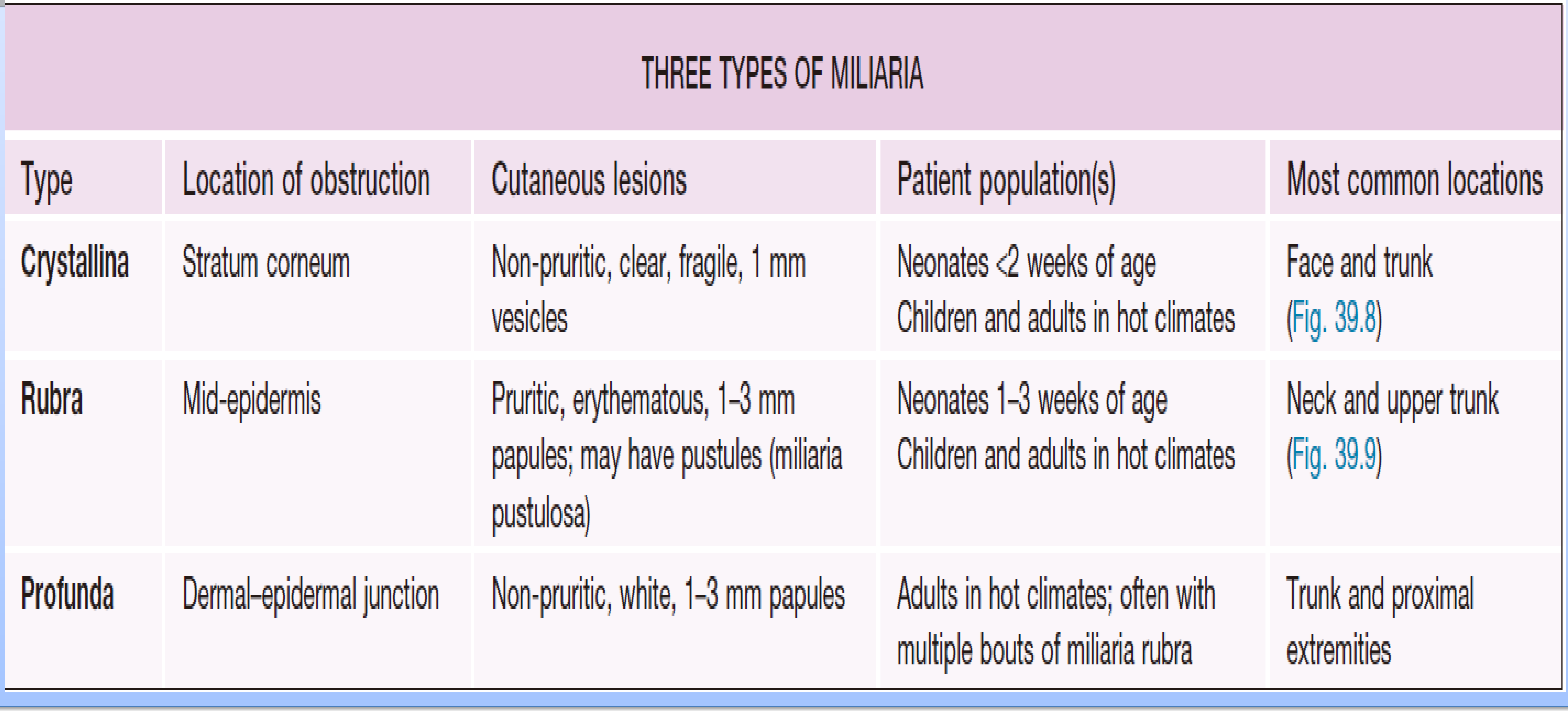
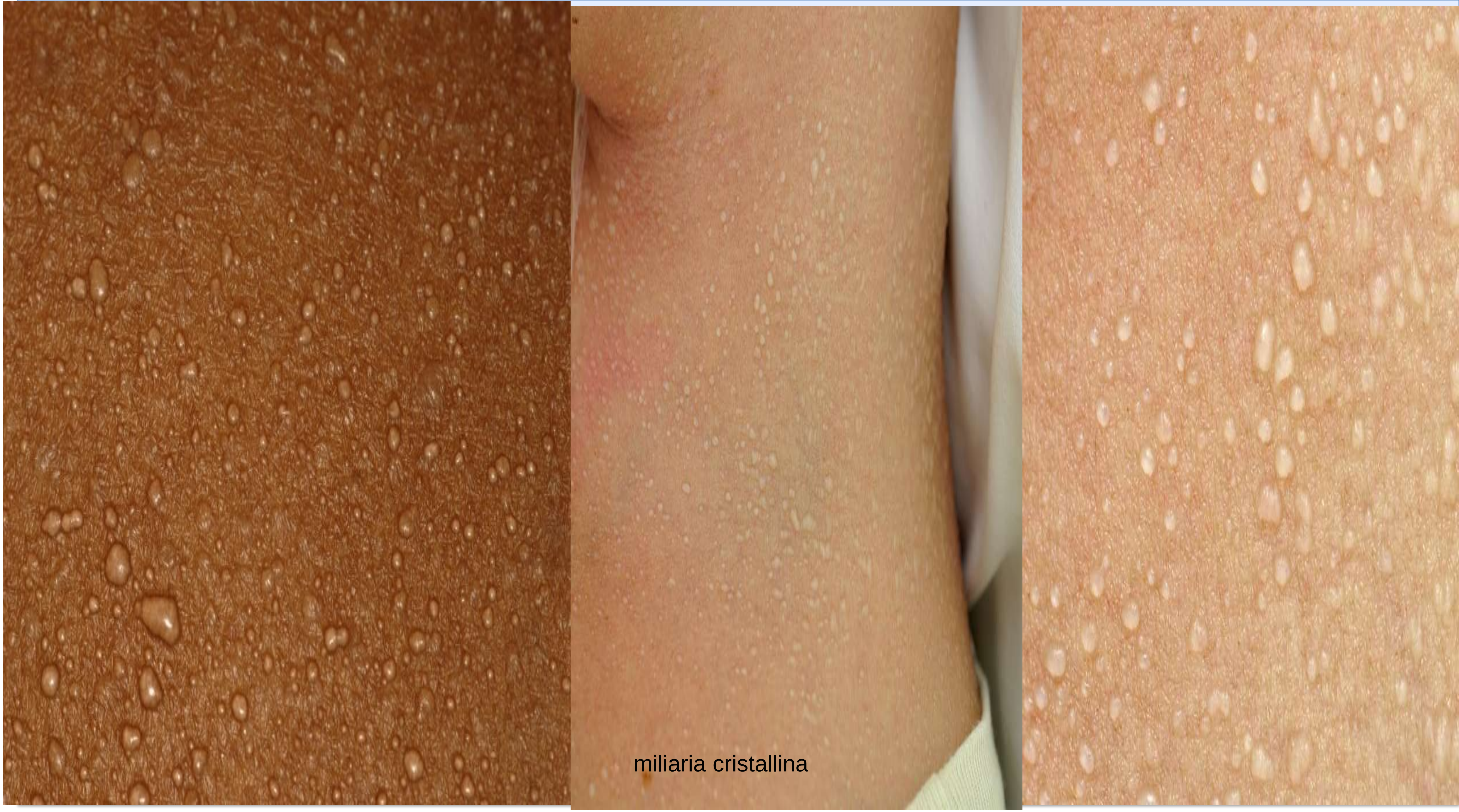
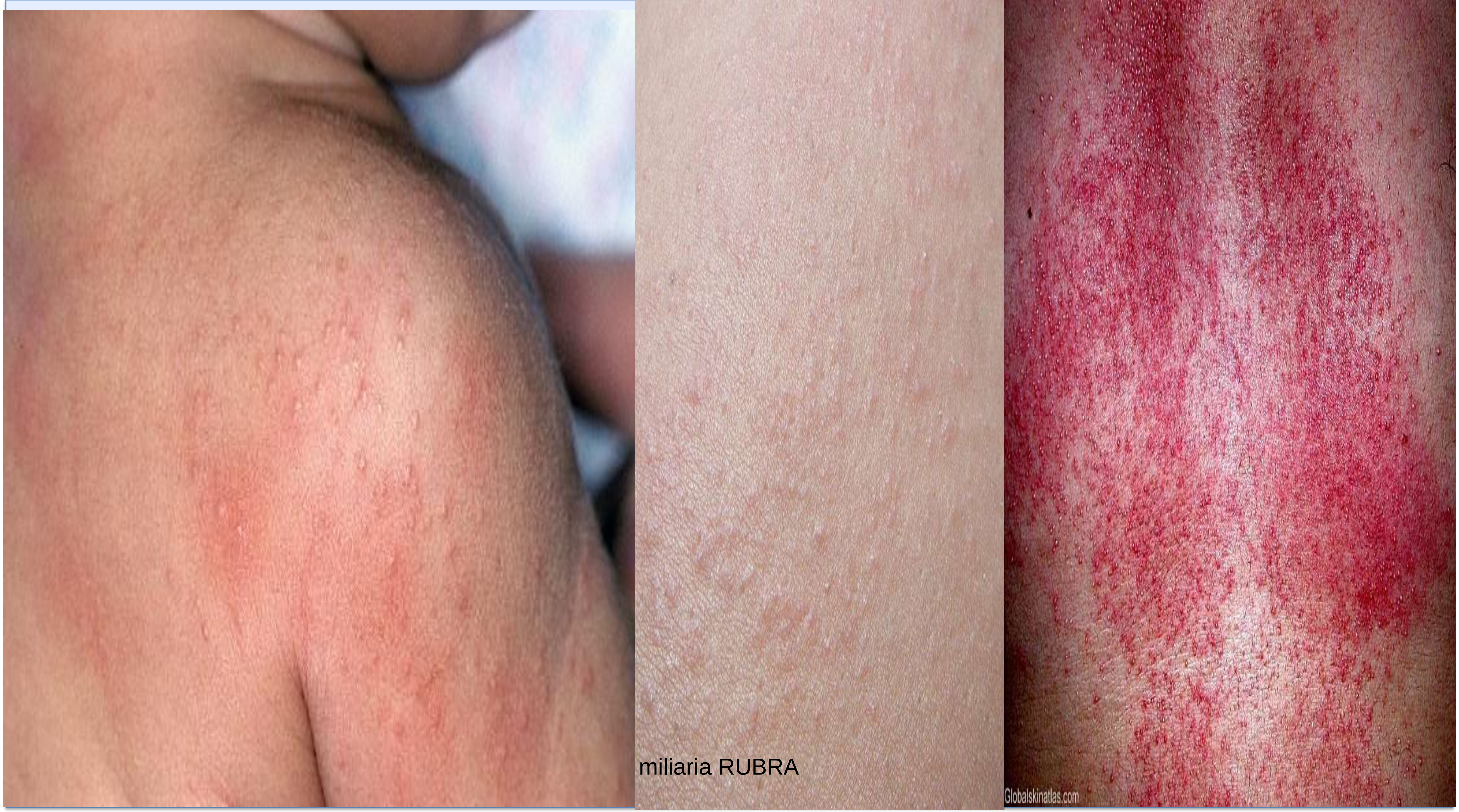


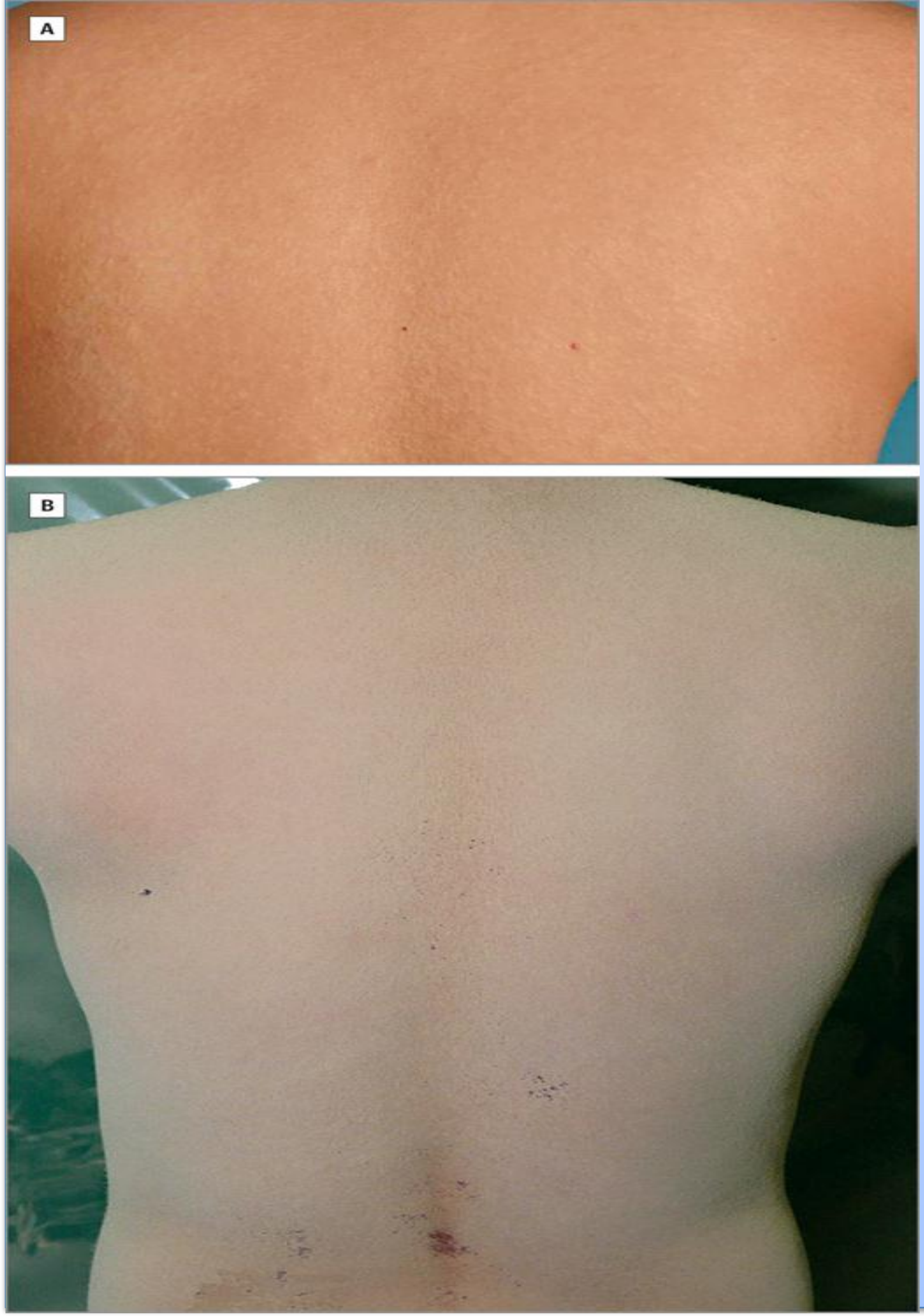
THREE TYPES OF MILIARIA				
Type	Location of obstruction	Cutaneous lesions	Patient population(s)	Most common locations
Crystallina	Stratum corneum	Non-pruritic, clear, fragile, 1 mm vesicles	Neonates <2 weeks of age Children and adults in hot climates	Face and trunk (Fig. 39.8)
Rubra	Mid-epidermis	Pruritic, erythematous, 1–3 mm papules; may have pustules (miliaria pustulosa)	Neonates 1–3 weeks of age Children and adults in hot climates	Neck and upper trunk (Fig. 39.9)
Profunda	Dermal–epidermal junction	Non-pruritic, white, 1–3 mm papules	Adults in hot climates; often with multiple bouts of miliaria rubra	Trunk and proximal extremities


miliaria cristallina

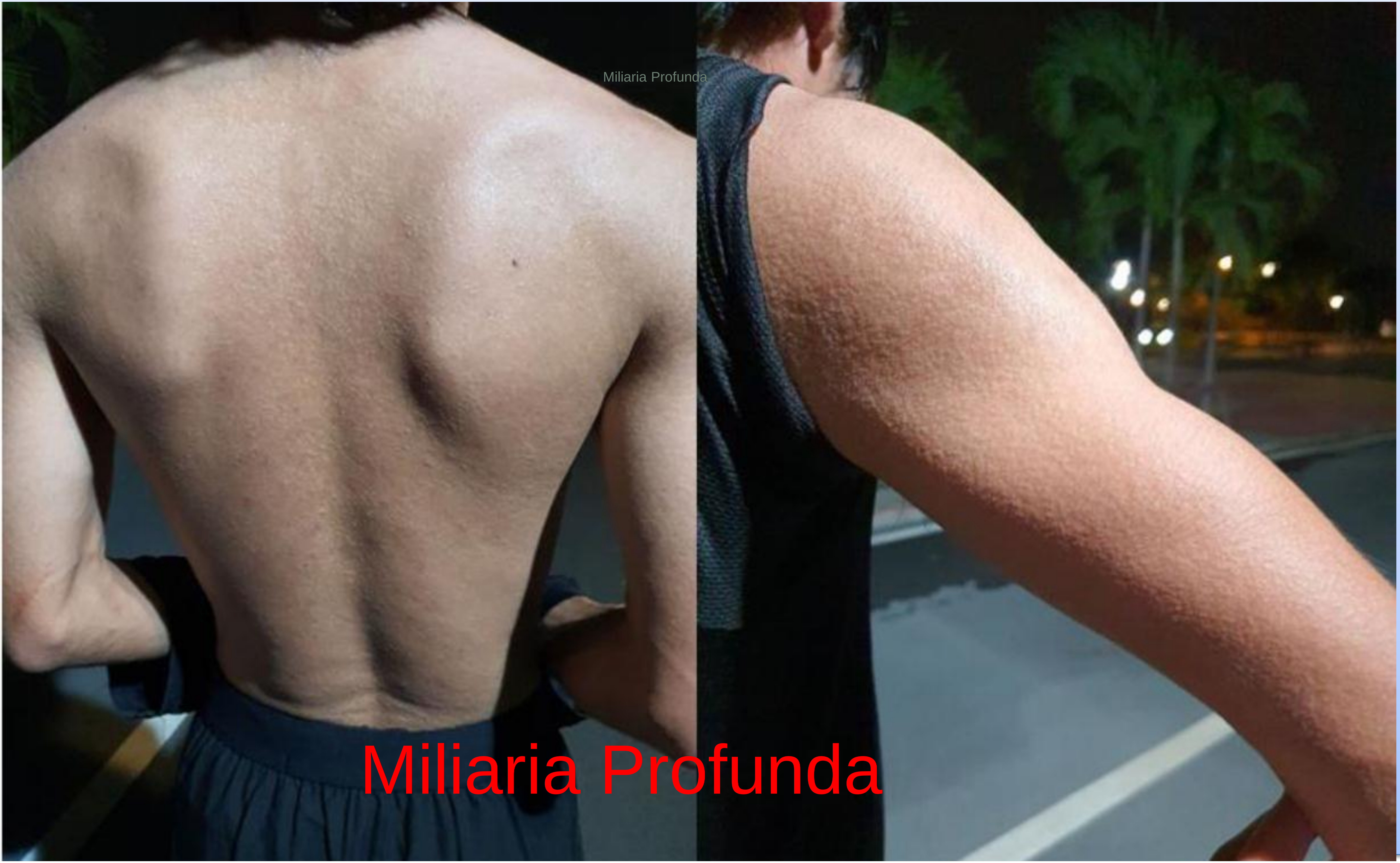


miliaria RUBRA

Globalskinatlas.com



A, Asymptomatic, nonfollicular whitish papules developed over the skin during active exercise. B, An admixture of starch-iodine powder was sprayed over the whole body after exercise using pressurized air through a spray gun, revealing generalized anhidrosis except for very limited areas (stained purple) on the lower back.


Miliaria Profunda

Miliaria Profunda

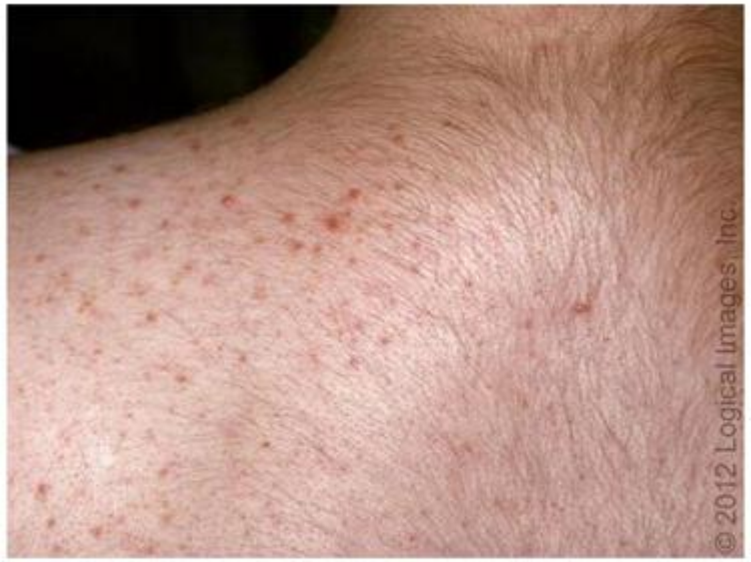
Diagnosis and differential diagnosis

miliaria

is diagnosed based on its typical clinical presentation.

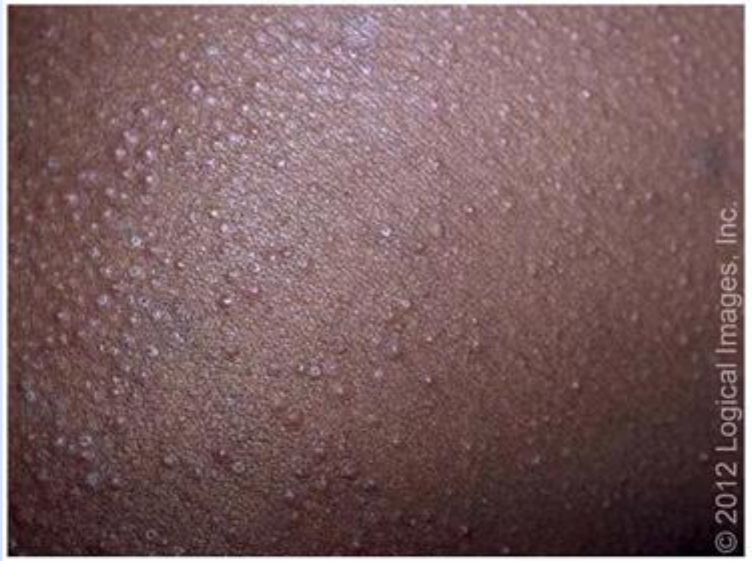
Differential diagnosis

***Pityrosporum* folliculitis**



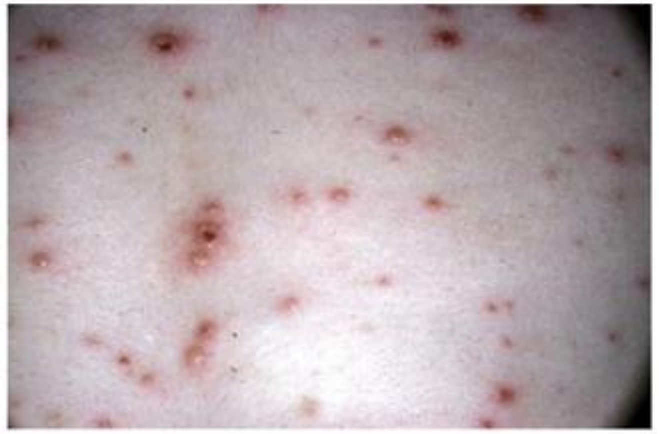
Follicular papules and pustules on the upper back of a patient with *Pityrosporum* folliculitis.

***Pityrosporum* folliculitis**



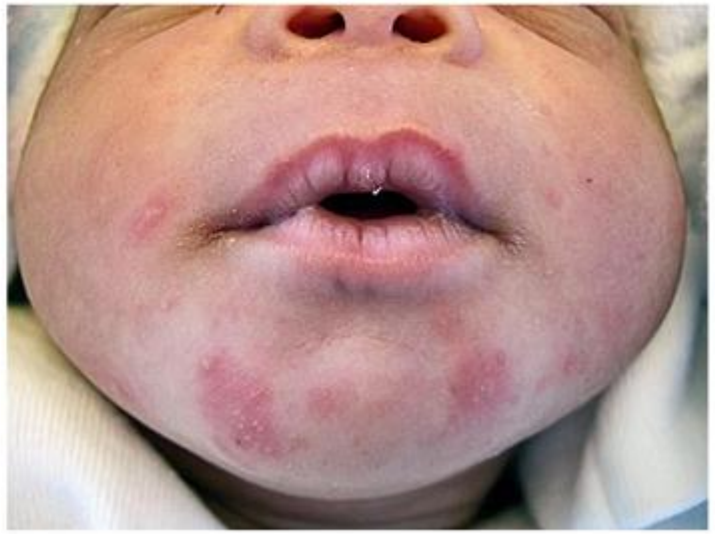
Pinpoint, follicular papules and pustules are characteristic of *Pityrosporum* folliculitis. The eruption most often involves the upper trunk.

Primary varicella lesions



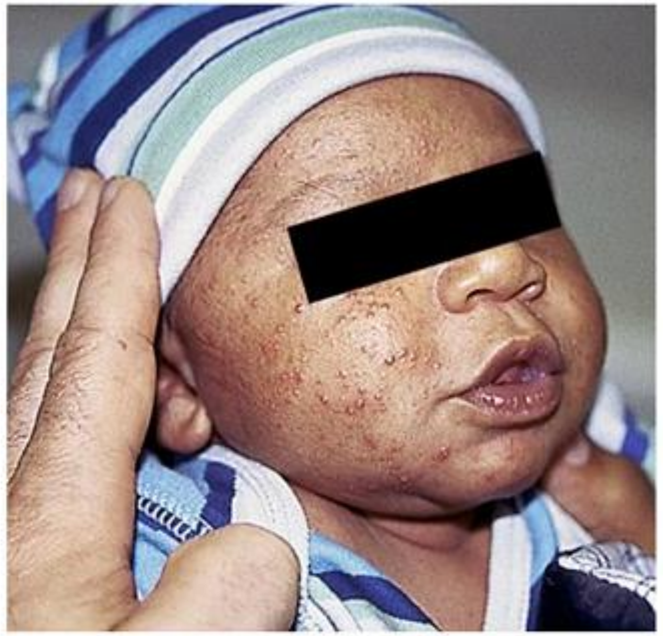
Vesicular lesions on an erythematous base are characteristic of chickenpox. The lesions occur in crops and are present in a variety of stages from maculopapular to vesicular or even pustular. Central necrosis and early crusting is also visible.

Erythema toxicum neonatorum



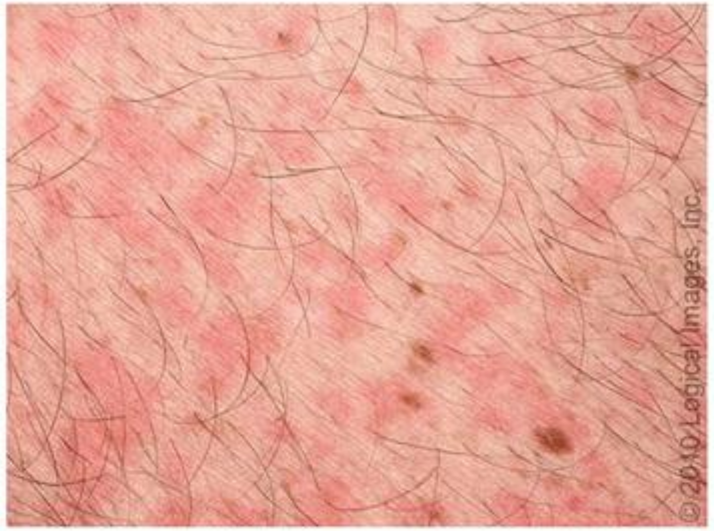
This healthy newborn developed a diffuse, transient eruption composed of red macules with central, pinpoint, whitish-yellow papules and pustules on the second day of life.

Neonatal acne



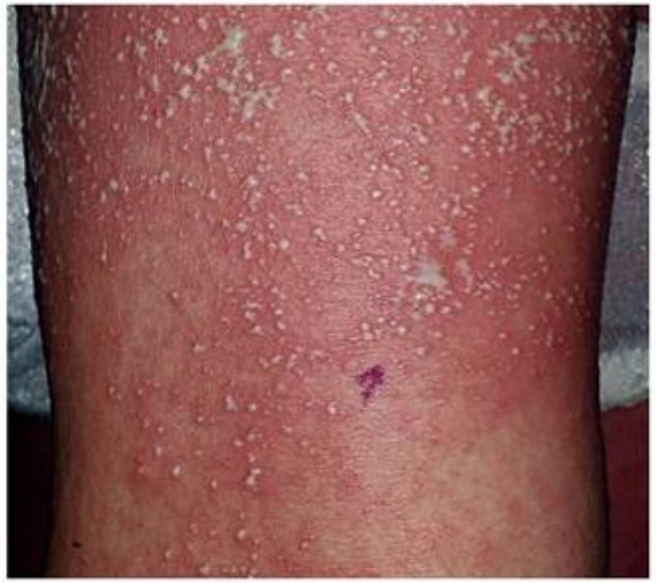
Erythematous papules and pustules on the cheeks of a 3-week-old neonate.

Viral exanthem



Multiple erythematous macules are present on the skin of this patient with a viral exanthem.

Acute generalized exanthematous pustulosis (AGEP)



Confluent nonfollicular pustules superimposed on edematous erythema in a 46-year-old woman with AGEP. A skin biopsy showed intracorneal pustules with numerous neutrophils and neutrophilic infiltration of the epidermis and upper dermis.

DISORDERS OF THE APOCRINE SWEAT GLAND

Fox–Fordyce Disease

Caused by plugging of the apocrine sweat glands
Occurs primarily in women 15–35 years of age
Skin-colored follicular papules develop in the axillary, anogenital, and periareolar areas
May be intensely pruritic

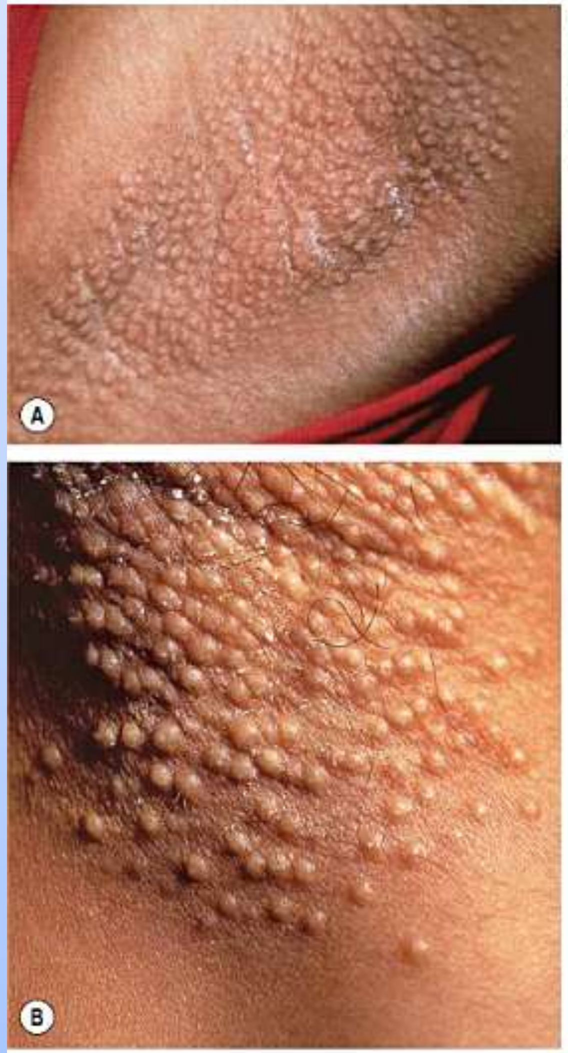


Fig. 39.14 Fox–Fordyce disease.
A,B Monomorphic skin-colored papules in the axillary vault. *Courtesy, Jean L Bolognia, MD.*

Treatment

There is no cure for Fox-Fordyce disease. Treatments include:

Topical retinoids
Topical steroids
Topical and oral antibiotics.

Bromhidrosis and Chromhidrosis

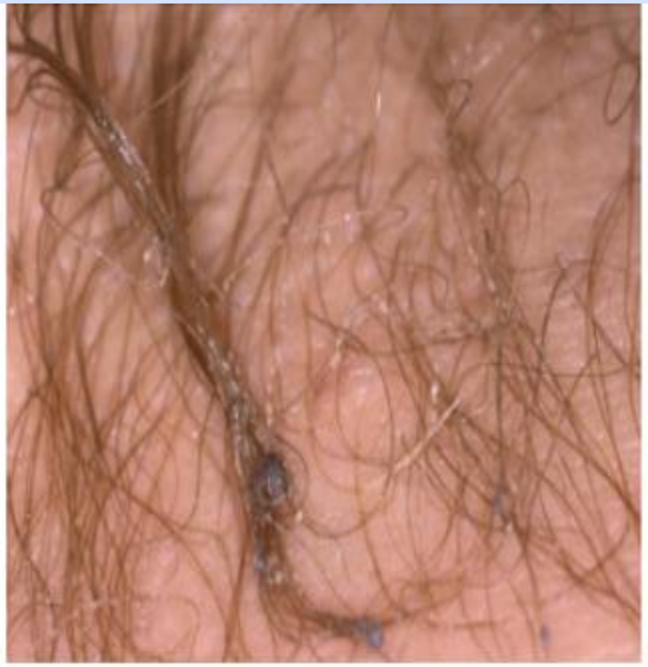
Bromhidrosis, abnormal sweat odour

Bromhidrosis, also known as osmidrosis or ozochrotia, is an unpleasant or offensive body odor due to any cause including:

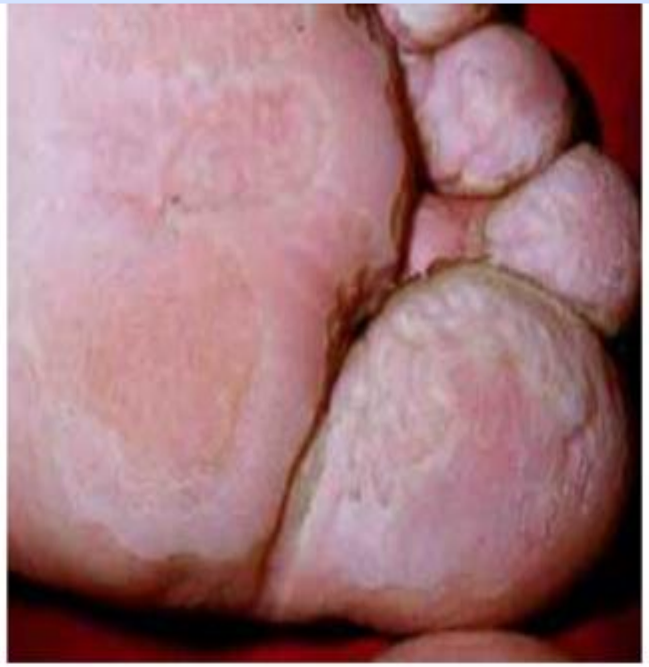
- Poor hygiene
- Infections
- Diet or medications, or inherited metabolic disorders

Bromhidrosis

Underarm sweating



Trichomycosis axillaris



Macerated skin over the toes and ball of the foot in pitted keratolysis

Bromhidrosis

Bromhidrosis may be classified as eccrine or apocrine, exogenous or endogenous:

➢ Exogenous

- Skin microbiota
- Skin infections — erythrasma, pitted keratolysis, trichomycosis, Sphingomonas paucimobilis.
- Nasal foreign body.

Bromhidrosis

Endogenous

- Foods ,Medications and toxins
- Systemic and skin disorders — obesity, diabetes mellitus, liver disease, renal failure, skin disorders of palms/soles and flexures.
- Metabolic disorders — trimethylaminuria, phenylketonuria, hypermethioninaemia.

Bromhidrosis

diagnosis and differential diagnosis

- Bromhidrosis is a clinical diagnosis
- Careful history and examination is required to determine the underlying cause.
- Investigations should be considered in children, if the smell is generalized also involving urine and breath, or if it is of recent onset unrelated to hygiene habits.

Differential diagnosis

- Body dysmorphophobia
- Olfactory hallucinations (due to neurological or psychiatric disorders)
- Olfactory refractory syndrome (fear of having an offensive body odor)

Bromhidrosis

What is the treatment for bromhidrosis?

1. General measures

- Good hygiene with regular washing using soaps and cleansers, deodorant, antiperspirant, removal of sweaty clothing
- Hair removal by shaving, electrolysis, or lasers
- Topical antiseptics
- Exfoliation of areas of keratin degradation

2. Specific measures

- Avoidance of odor-causing foods and medications
- Treatment of skin infections
- Treatment of hyperhidrosis
- Removal of nasal foreign body
- Removal of apocrine glands — laser, surgery, liposuction curettage, microwa induced fibrosis
- Dietary modification for metabolic disorders

Chromhidrosis

Chromhidrosis

- In the normal state, sweat is colorless.

The term "chromhidrosis," derived from the Greek "chroma" (colored) and "hidros" (sweat), describes the occurrence of colored sweat.

Chromhidrosis

- **True chromhidrosis** is a rare condition characterized by the secretion of colored sweat from apocrine or eccrine sweat glands.

- **In pseudochromhidrosis**, a far more common disorder, sweat becomes colored after secretion from the sweat gland. Pseudochromhidrosis results from contact between sweat and dyes, chemicals, or chromogenic bacteria on the skin.

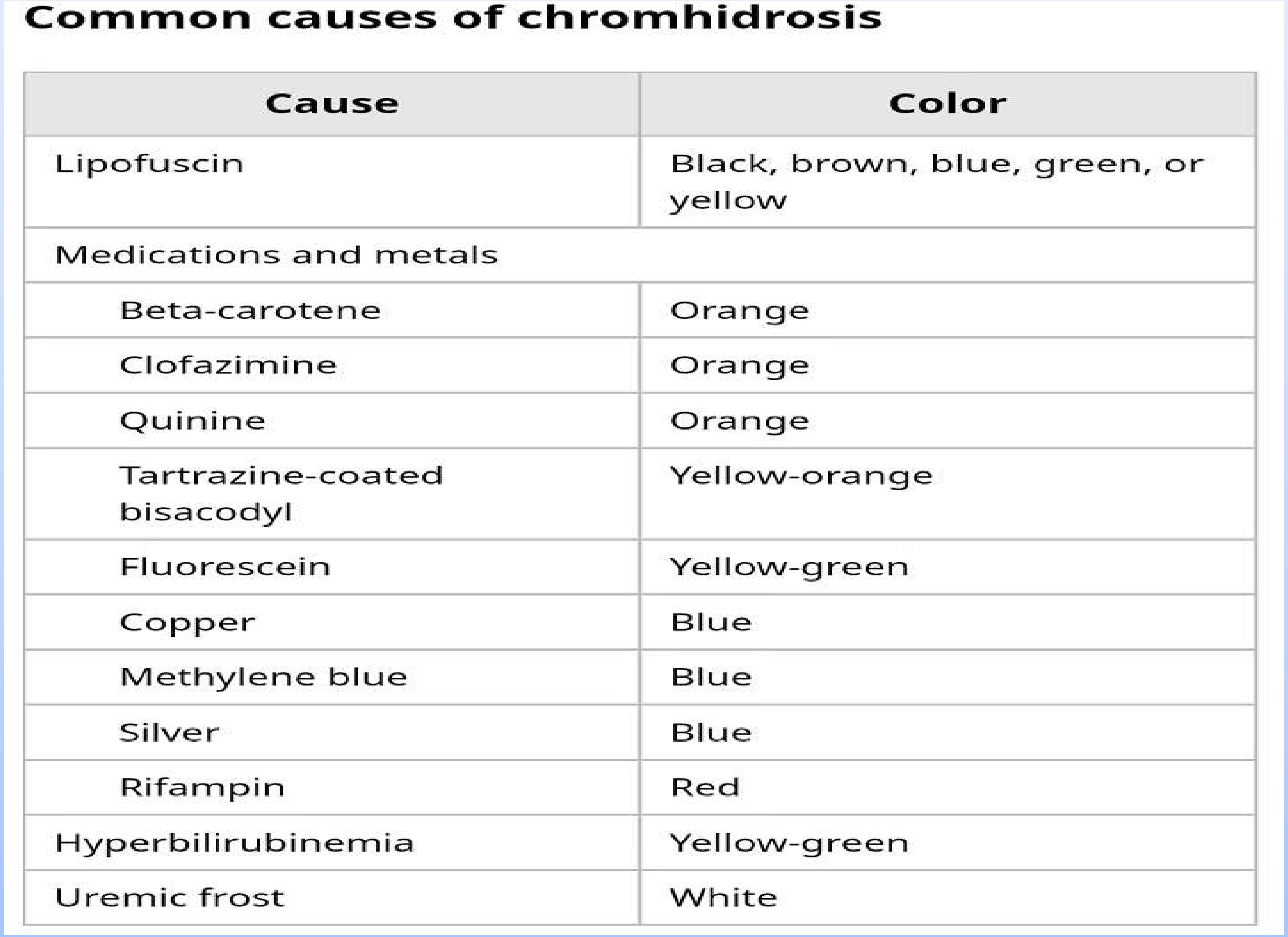
Chromhidrosis

Etiology

- **Apocrine chromhidrosis** is most often due to the presence of lipofuscin granules
- Lipofuscin is a yellowish pigment that is thought to be produced by the oxidation of unsaturated fatty acids.
- The color of secreted sweat is determined by the oxidation state of the lipofuscin granules; sweat can be black, brown, blue, green, yellow, or pink . The higher the state of oxidation, the darker the color appears

Chromhidrosis

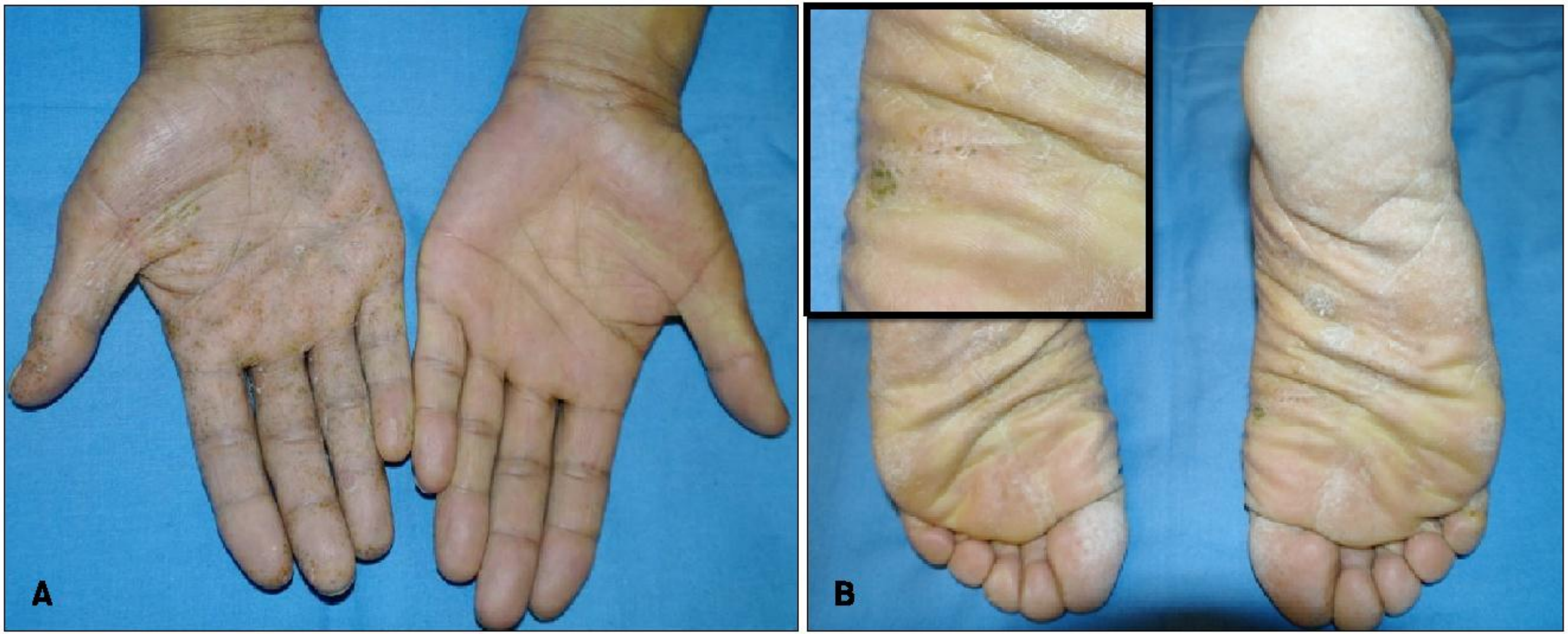
- **Eccrine chromhidrosis** has multiple causes.
- lipofuscin granules can be secreted by the eccrine sweat gland
- systemic medications, heavy metals, or other agents can be secreted in sweat causing discoloration of the sweat, Hyperbilirubinemia is a rare cause of eccrine chromhidrosis



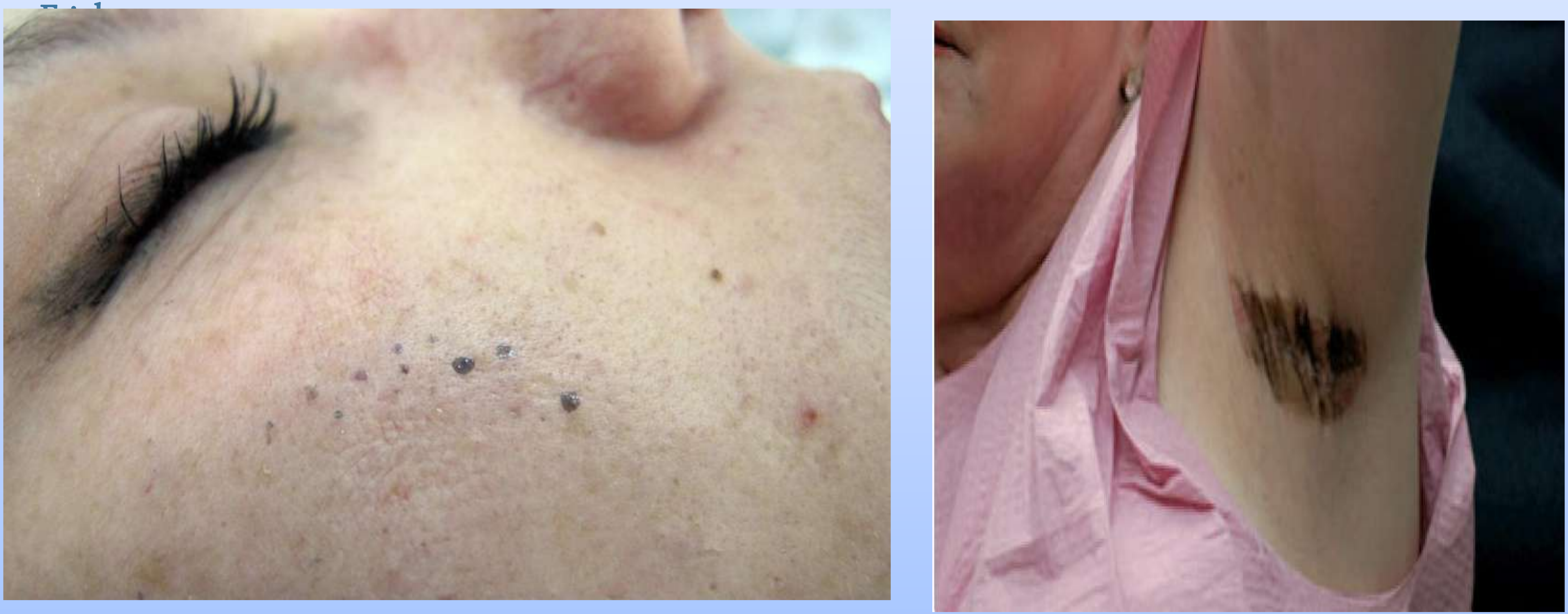
Common causes of chromhidrosis

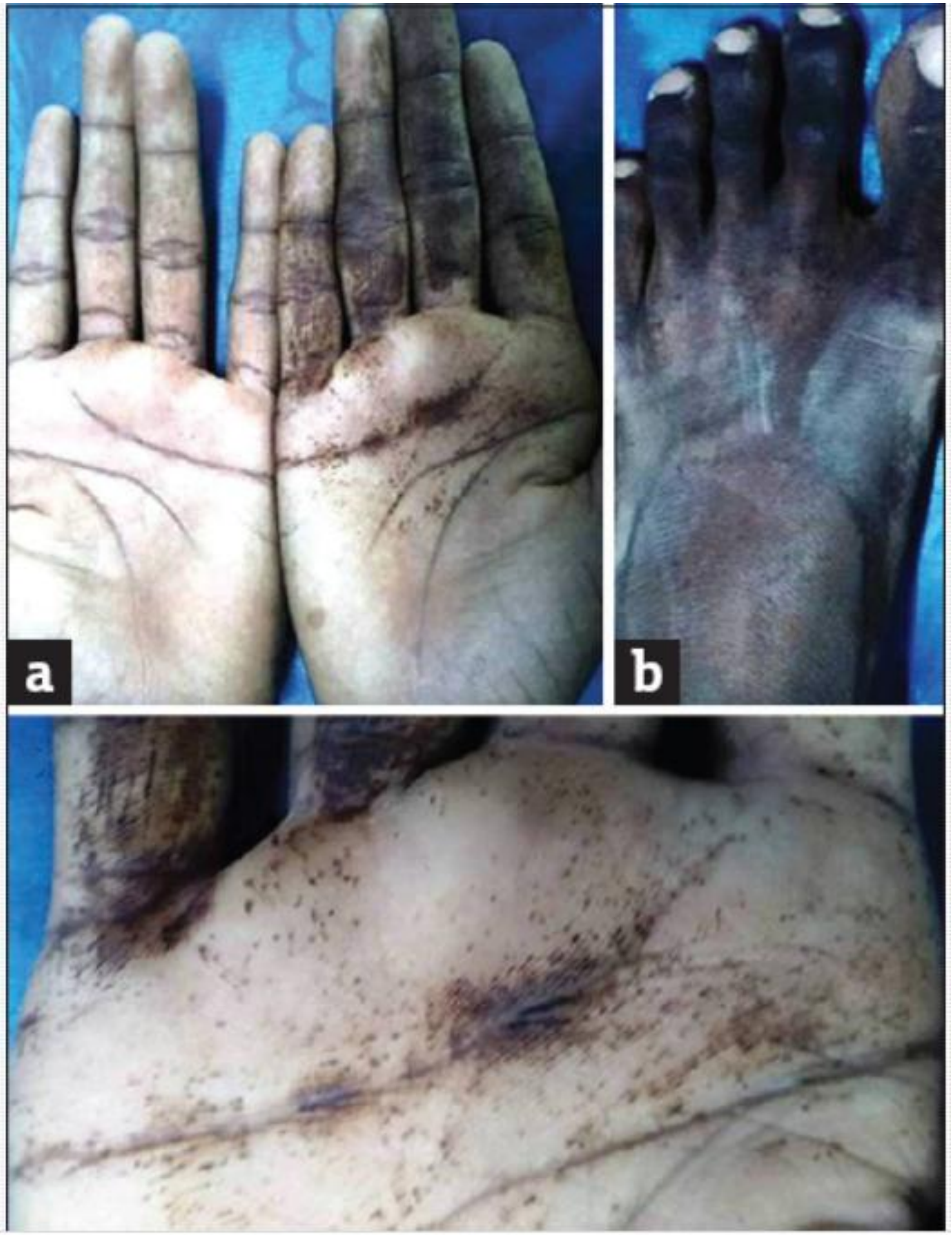
Cause	Color
Lipofuscin	Black, brown, blue, green, or yellow
Medications and metals	
Beta-carotene	Orange
Clofazimine	Orange
Quinine	Orange
Tartrazine-coated bisacodyl	Yellow-orange
Fluorescein	Yellow-green
Copper	Blue
Methylene blue	Blue
Silver	Blue
Rifampin	Red
Hyperbilirubinemia	Yellow-green
Uremic frost	White

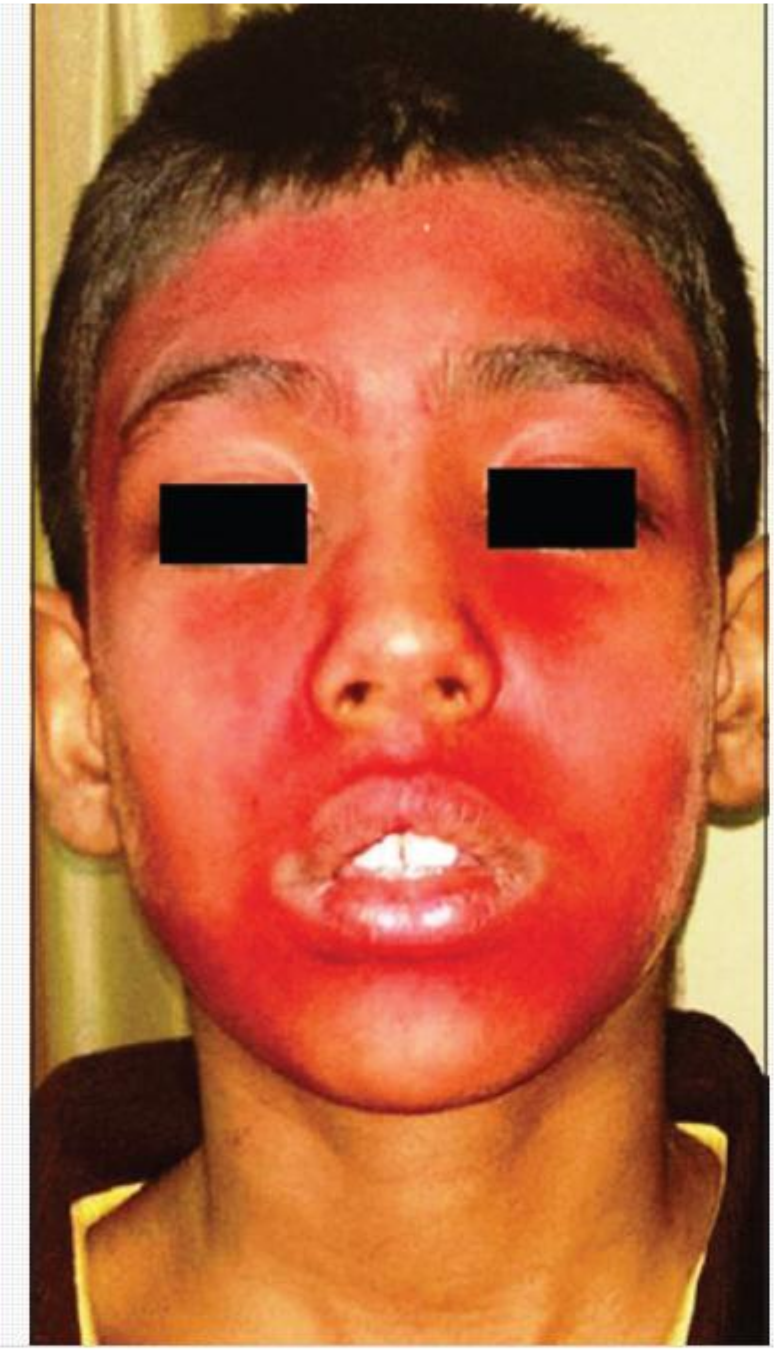
Chromhidrosis



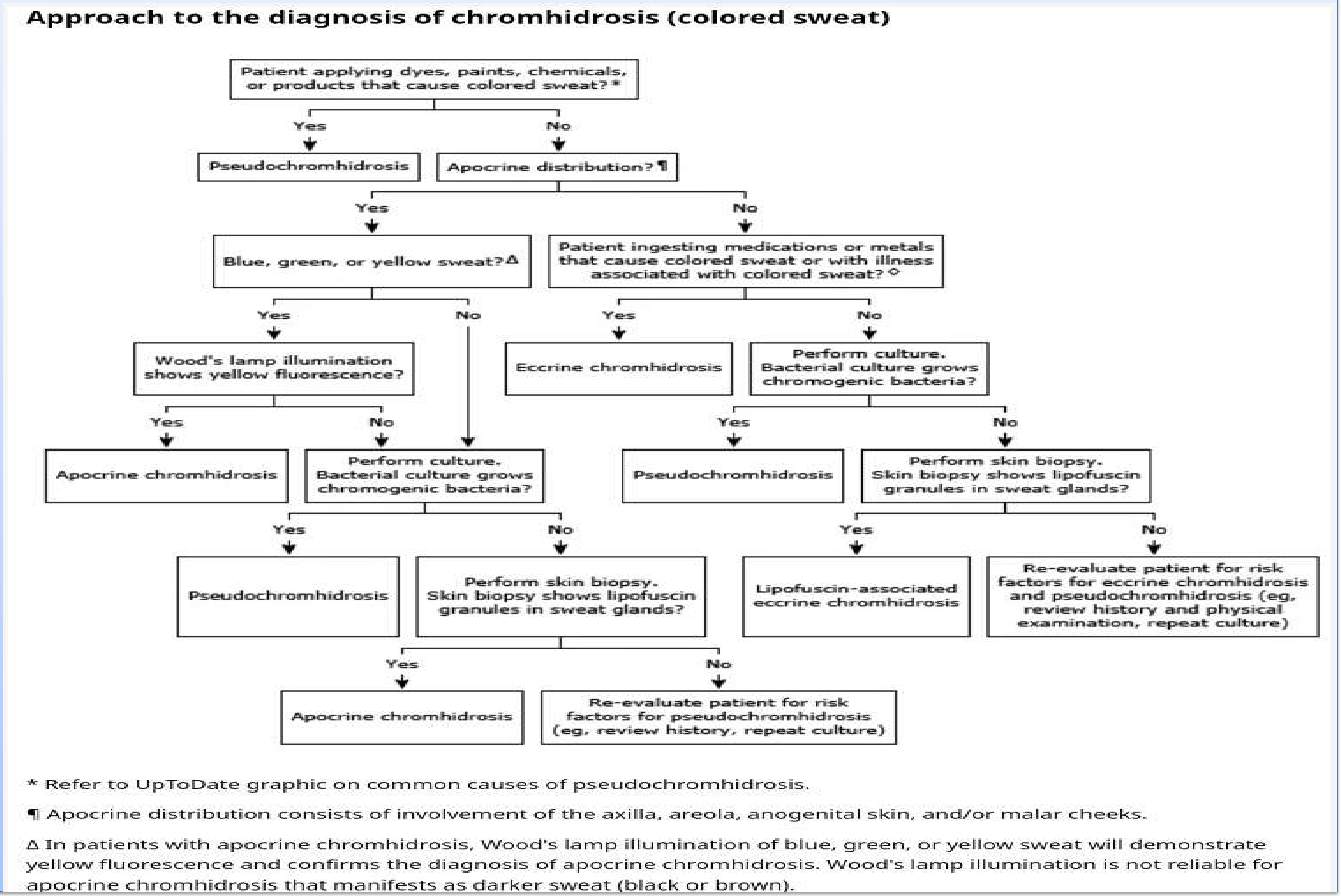
Chromhidrosis




a
b



Diagnosis



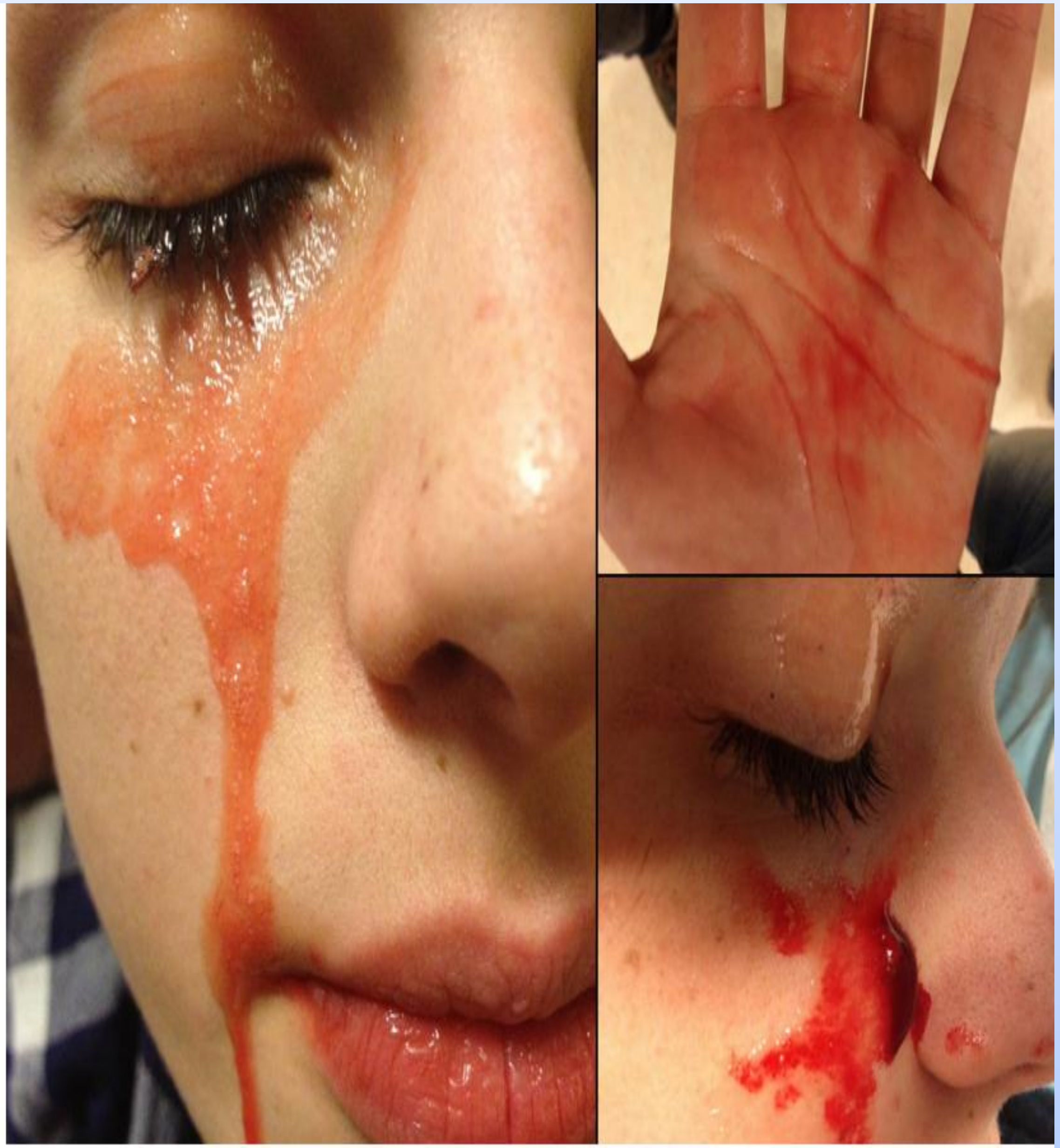
Approach to the diagnosis of chromhidrosis (colored sweat)

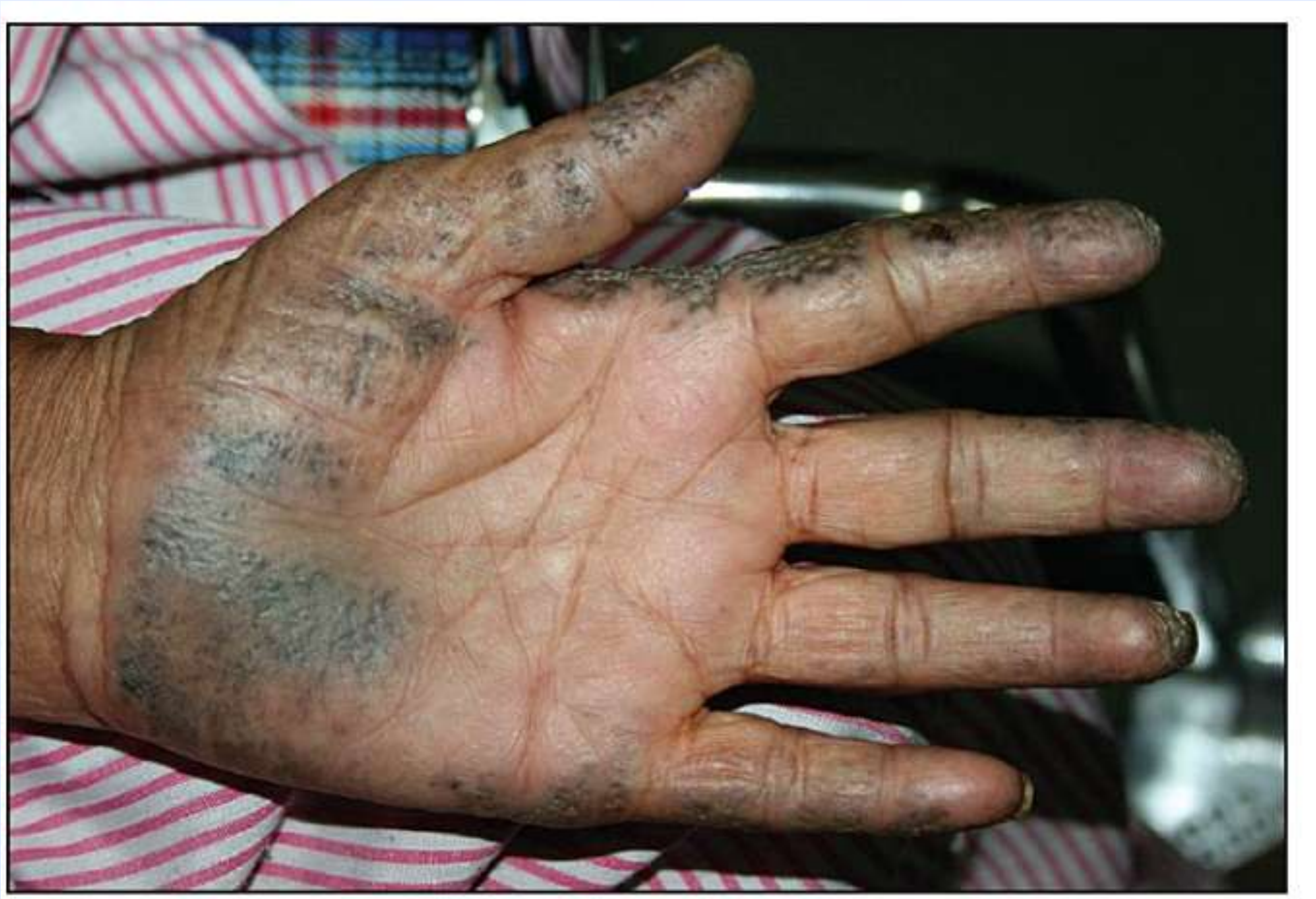
- Patient applying dyes, paints, chemicals, or products that cause colored sweat?*
  - Yes → Pseudochromhidrosis
  - No → Apocrine distribution?¶
    - Yes → Blue, green, or yellow sweat?Δ
      - Yes → Wood's lamp illumination shows yellow fluorescence?
        - Yes → Apocrine chromhidrosis
        - No → Perform culture. Bacterial culture grows chromogenic bacteria?
      - No → Perform culture. Bacterial culture grows chromogenic bacteria?
        - Yes → Pseudochromhidrosis
        - No → Perform skin biopsy. Skin biopsy shows lipofuscin granules in sweat glands?
          - Yes → Apocrine chromhidrosis
          - No → Re-evaluate patient for risk factors for pseudochromhidrosis (eg, review history, repeat culture)
    - No → Patient ingesting medications or metals that cause colored sweat or with illness associated with colored sweat?◊
      - Yes → Eccrine chromhidrosis
      - No → Perform culture. Bacterial culture grows chromogenic bacteria?
        - Yes → Pseudochromhidrosis
        - No → Perform skin biopsy. Skin biopsy shows lipofuscin granules in sweat glands?
          - Yes → Lipofuscin-associated eccrine chromhidrosis
          - No → Re-evaluate patient for risk factors for eccrine chromhidrosis and pseudochromhidrosis (eg, review history and physical examination, repeat culture)

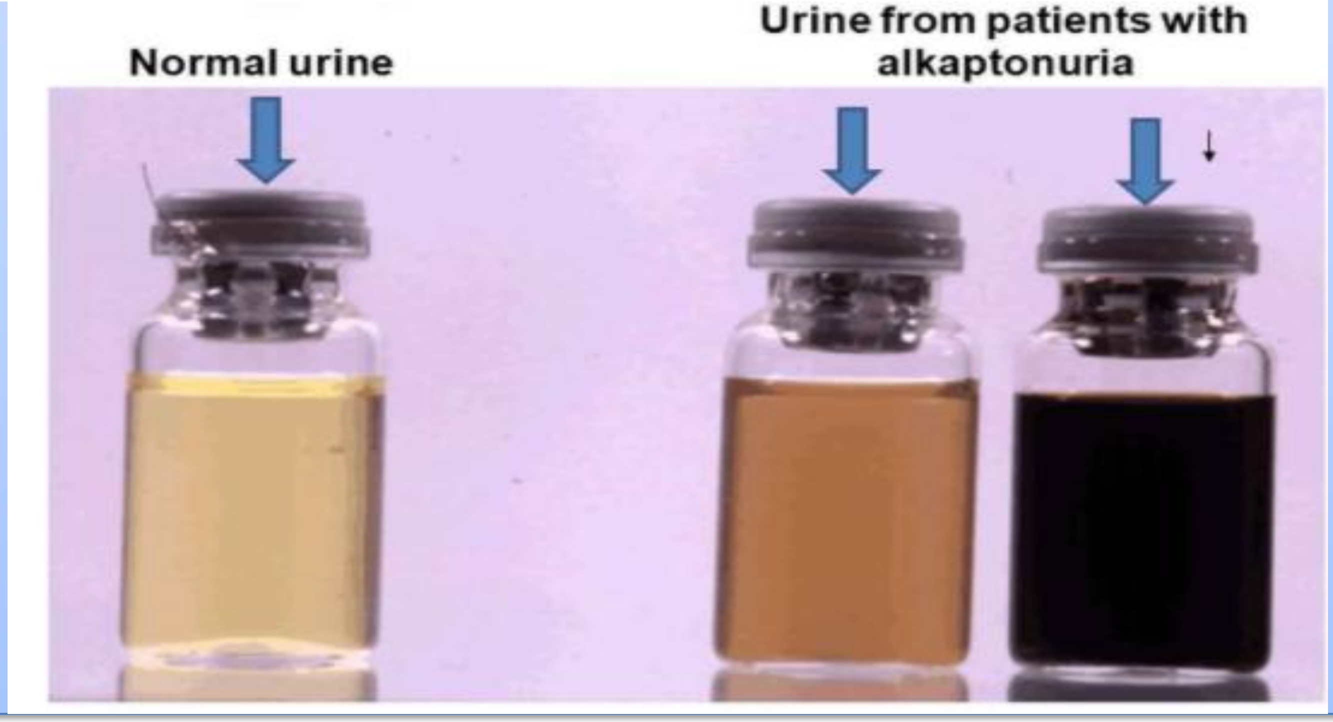
* Refer to UpToDate graphic on common causes of pseudochromhidrosis.

¶ Apocrine distribution consists of involvement of the axilla, areola, anogenital skin, and/or malar cheeks.

Δ In patients with apocrine chromhidrosis, Wood's lamp illumination of blue, green, or yellow sweat will demonstrate yellow fluorescence and confirms the diagnosis of apocrine chromhidrosis. Wood's lamp illumination is not reliable for apocrine chromhidrosis that manifests as darker sweat (black or brown).






Normal urine
Urine from patients with alkaptonuria

Chromhidrosis

DIFFERENTIAL DIAGNOSIS

- Differential diagnosis for chromhidrosis and pseudochromhidrosis includes :
- alkaptonuria
- Hematidrosis
- Disorders associated with cutaneous hyperpigmentation:

Chromhidrosis

Treatment

- Data on the treatment of chromhidrosis and pseudochromhidrosis are limited.
- For patients with apocrine chromhidrosis, we suggest topical capsaicin as initial treatment
- Injection of botulinum toxin into the affected area is an alternative treatment.

Chromhidrosis

Treatment

- Other treatments to inhibit sweating (eg, iontophoresis, oral anticholinergics, surgical removal of sweat glands, laser therapy) have also been proposed as treatment options; however, efficacy for chromhidrosis is unclear.

Chromhidrosis

Treatment

- Patients with an identifiable underlying cause of chromhidrosis or pseudochromhidrosis (eg, chromogenic bacteria) may experience resolution of symptoms following elimination of the inciting factor, exposure, or illness.

For more Question feel free to contact me

mobile: +967778444430

Email : almurieshmaher@gmail.com

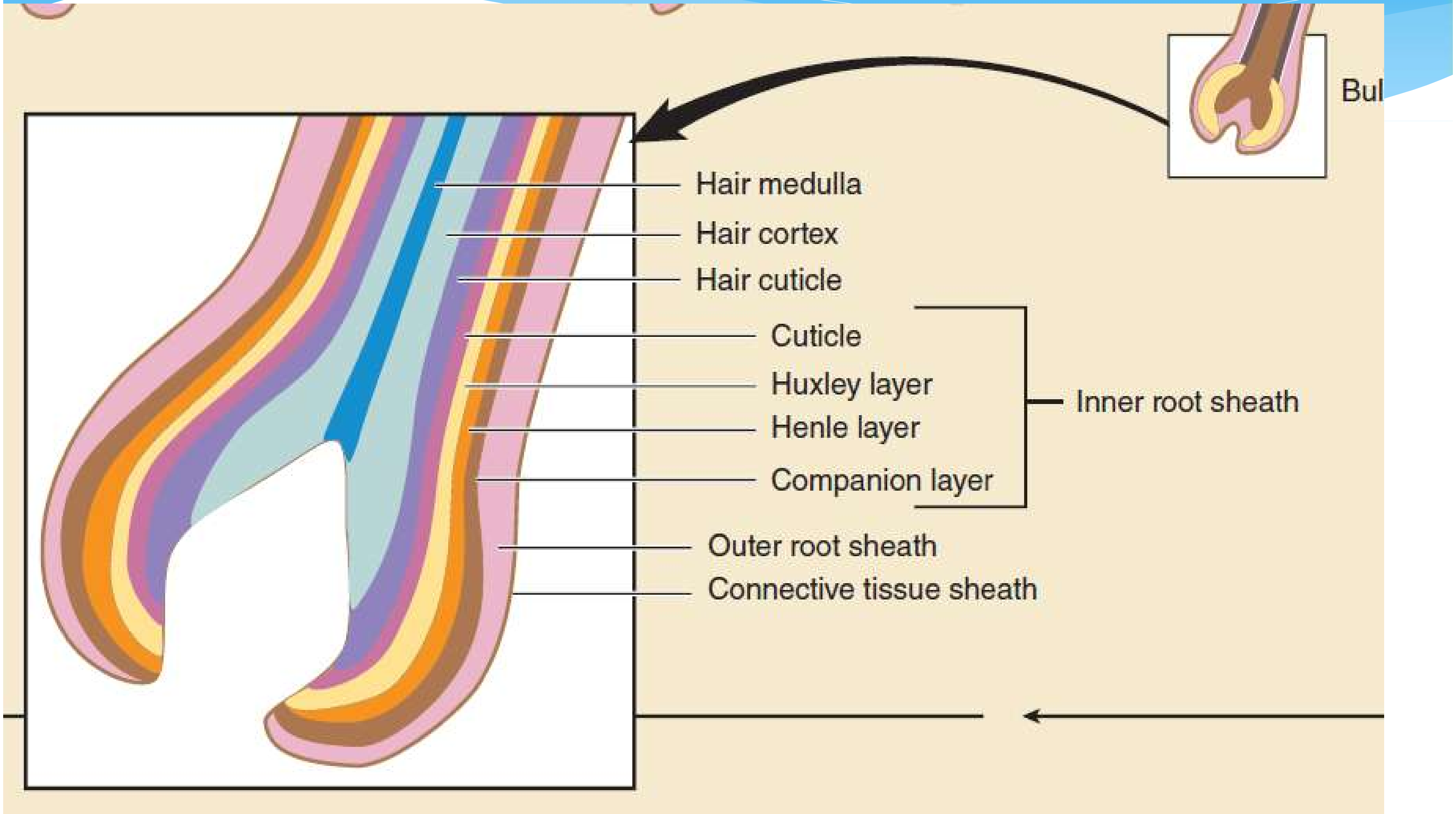
Hair Disorder

ASST. PROF. AL-MURIESH MAHER , MBBS, MSc, M.D

Scope of the lecture

- Hair anatomy
- Outline the definition alopecia
- Clinical approach to alopecia
- Alopecia (non cicatricial)
- Alopecia (cicatricial)
- Review of hypertrichosis.
- Summarize the treatment options for hypertrichosis.
- Hirsutism
- Treatment of Hrsutism

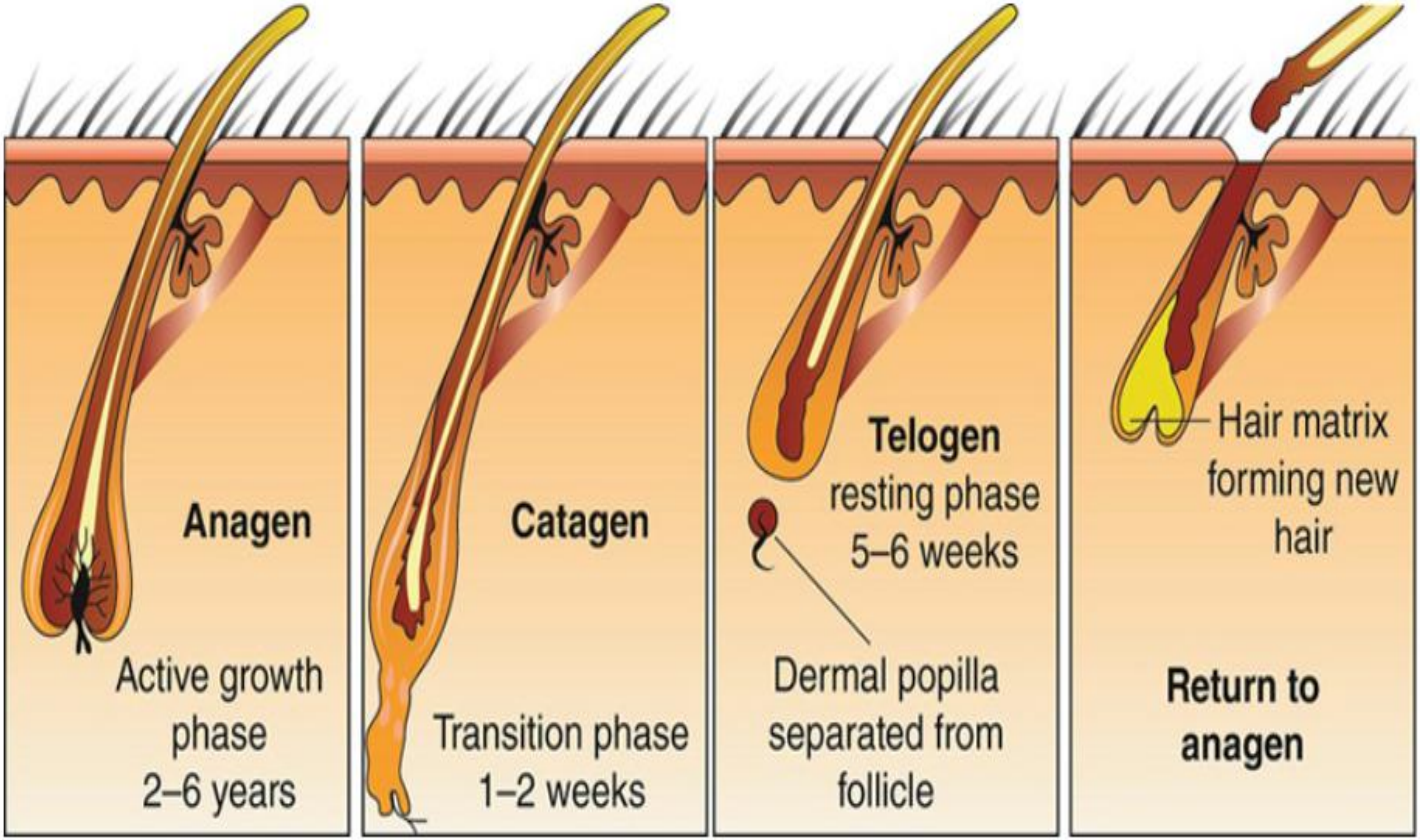
hair anatomy


Bul
Hair medulla
Hair cortex
Hair cuticle
Cuticle
Huxley layer
Henle layer
Companion layer
Inner root sheath
Outer root sheath
Connective tissue sheath

Alopecia

- The term alopecia means hair loss regardless of the cause
- Hundreds of thousands of hair on the head.
- The hair cycle consists of three phases:

the growth phase	(**anagen**)
the resting phase	(**catagen**)
the shedding phase	(**telogen**)


Anagen
Active growth phase 2–6 years
Catagen
Transition phase 1–2 weeks
Telogen
resting phase 5–6 weeks
Dermal popilla separated from follicle
Hair matrix forming new hair
Return to anagen

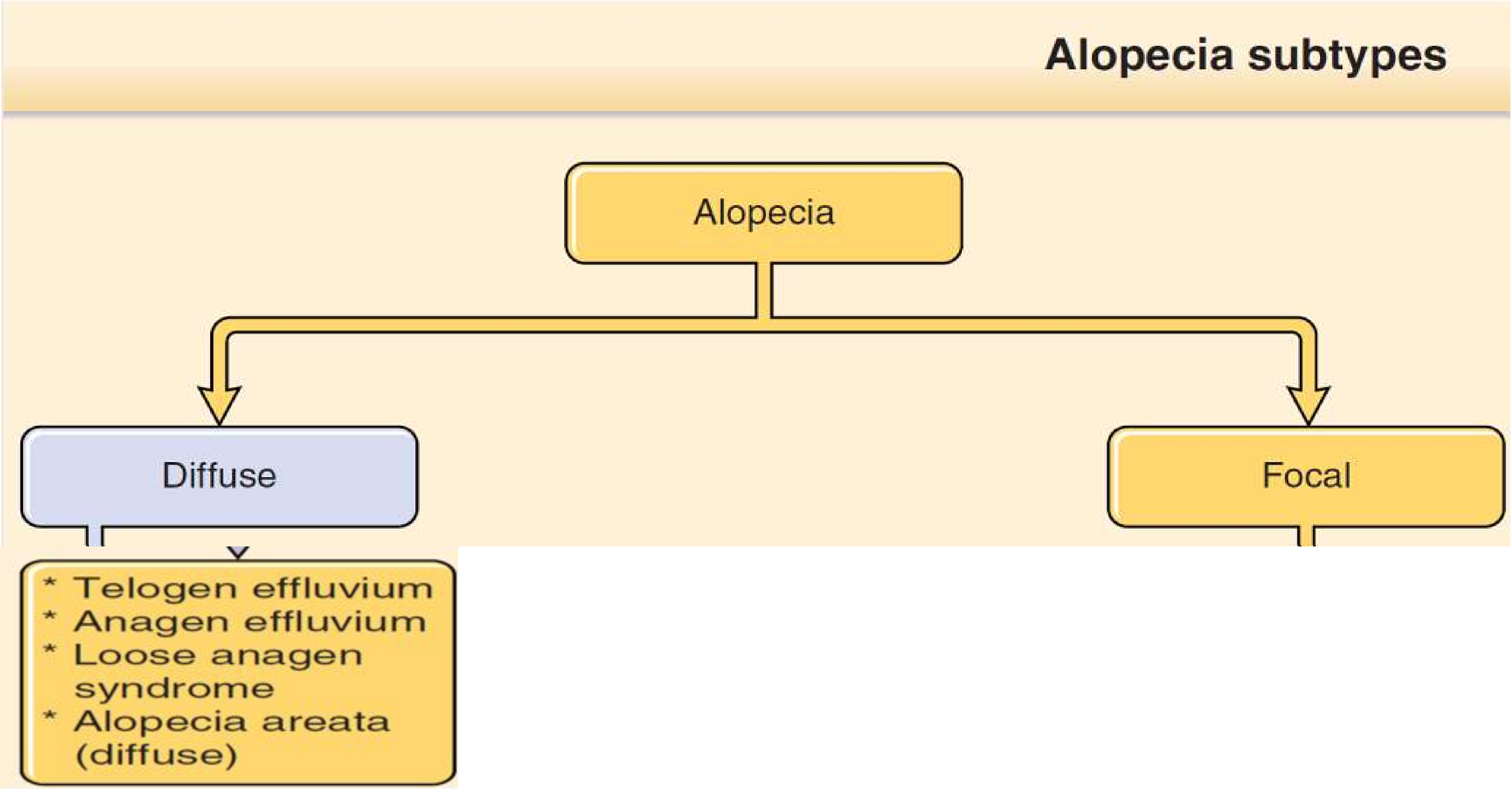
Natural growth and fall cycles of scalp hair.

Alopecia

90%of hair are in the anagen phase and 10% in resting and shedding phases.

- Alopecia can be subdivided into two main subtype : **diffuse and focal**

Alopecia subtype



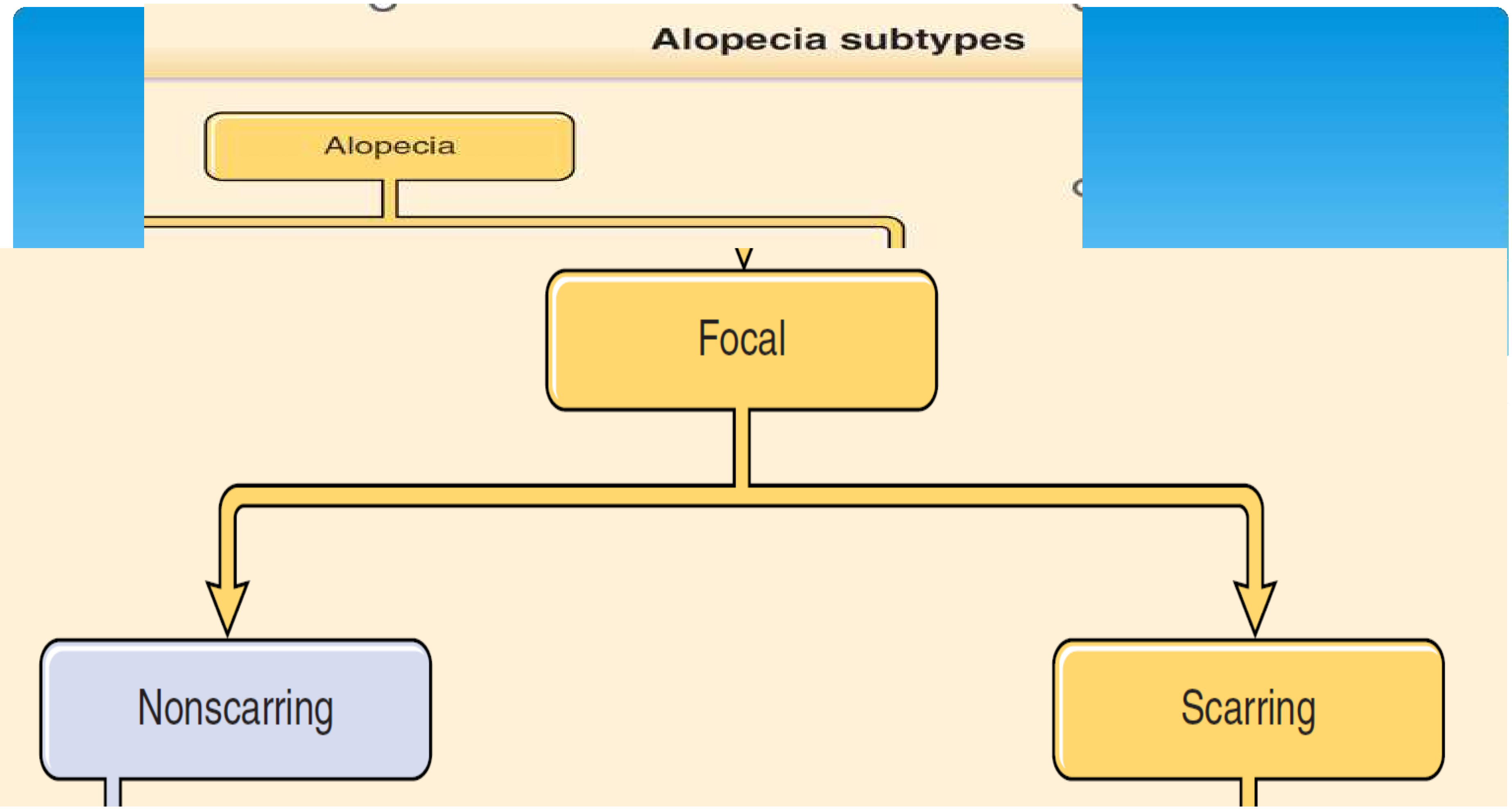
Alopecia subtypes

Alopecia

Diffuse

Focal

- * Telogen effluvium
- * Anagen effluvium
- * Loose anagen syndrome
- * Alopecia areata (diffuse)



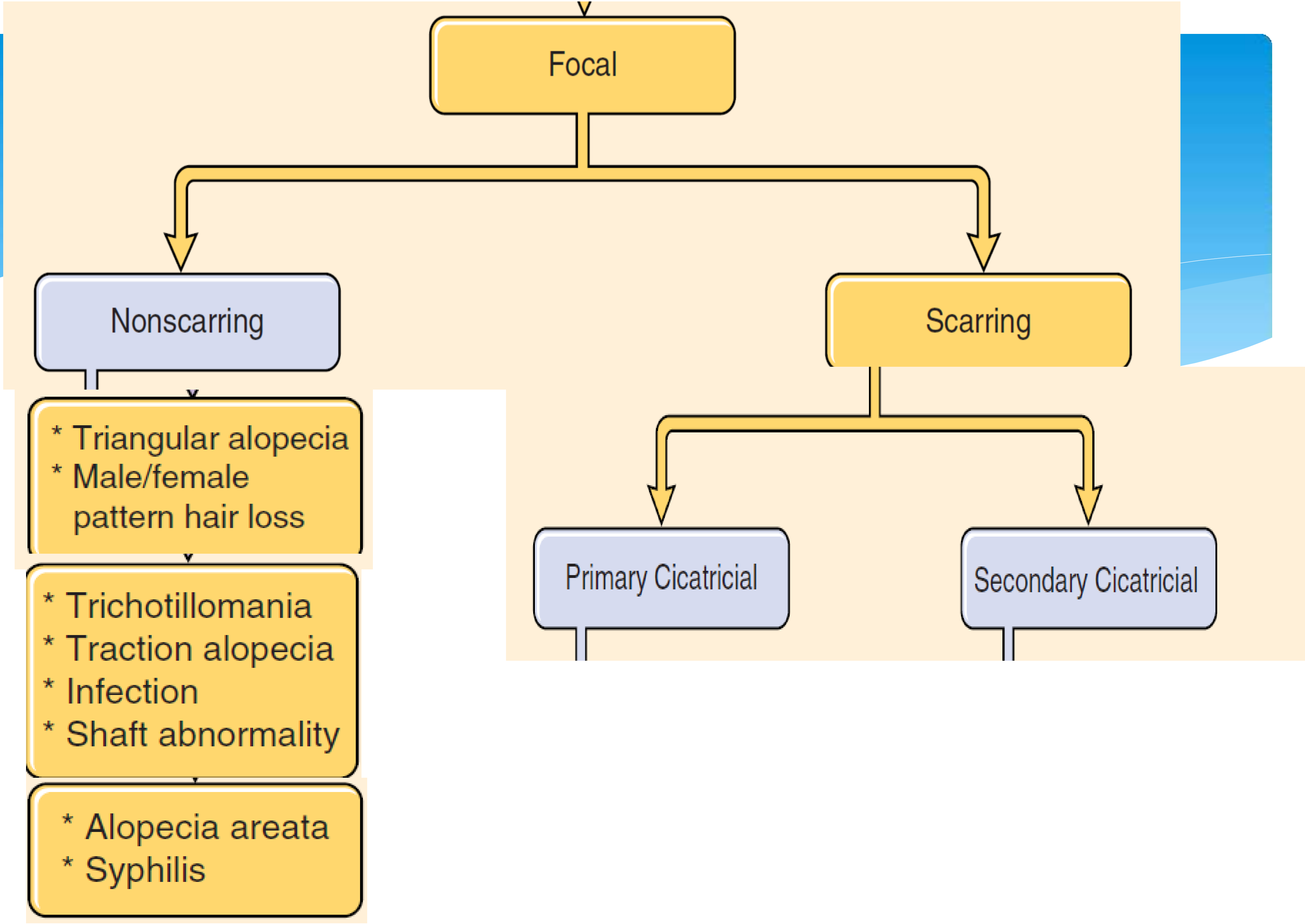
Alopecia subtypes

Alopecia

Focal

Nonscarring

Scarring



Focal

Nonscarring

- * Triangular alopecia
- * Male/female pattern hair loss

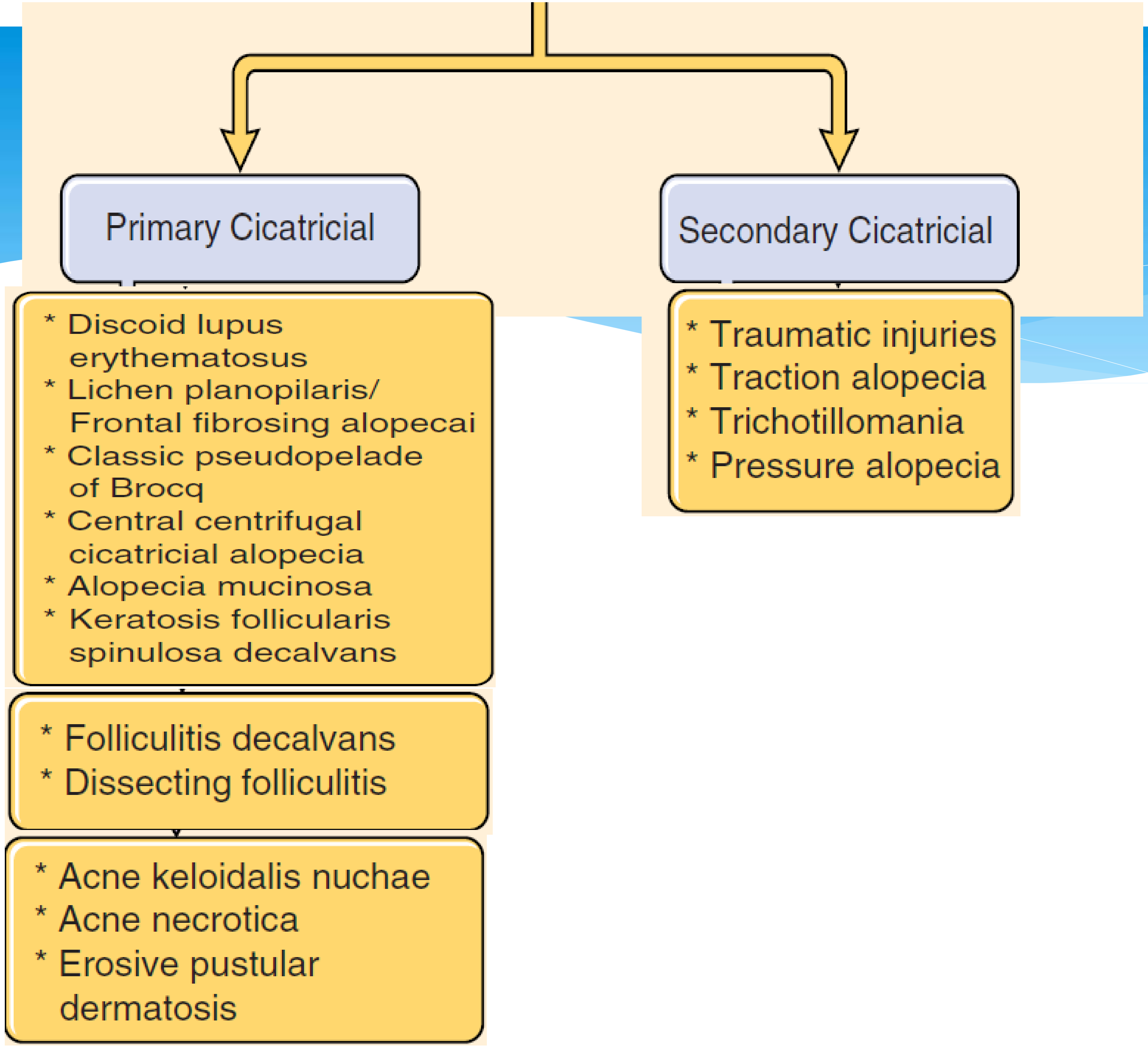
- * Trichotillomania
- * Traction alopecia
- * Infection
- * Shaft abnormality

- * Alopecia areata
- * Syphilis

Scarring

Primary Cicatricial

Secondary Cicatricial



Primary Cicatricial

- Discoid lupus erythematosus
- Lichen planopilaris/ Frontal fibrosing alopecai
- Classic pseudopelade of Brocq
- Central centrifugal cicatricial alopecia
- Alopecia mucinosa
- Keratosis follicularis spinulosa decalvans

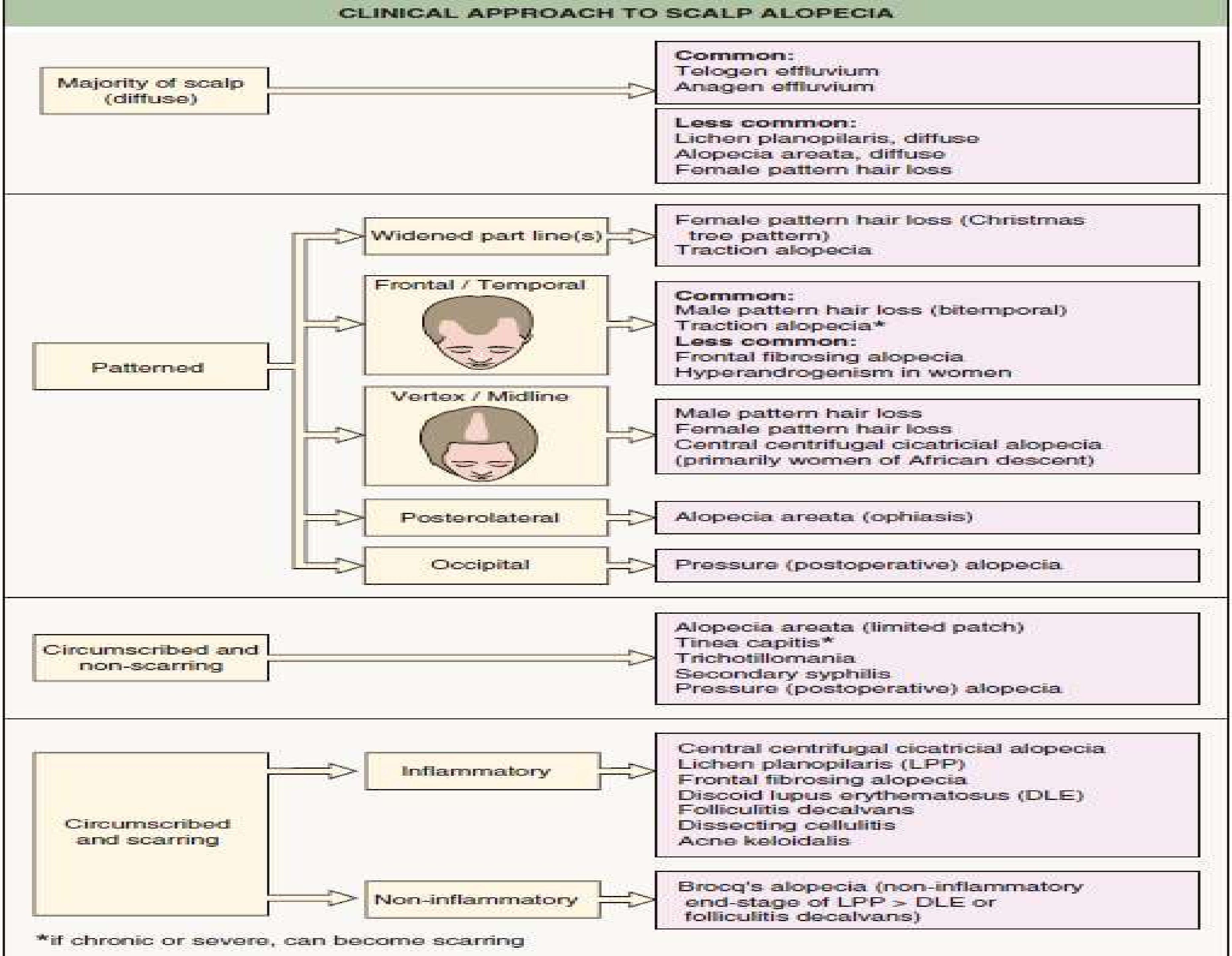
- Folliculitis decalvans
- Dissecting folliculitis

- Acne keloidalis nuchae
- Acne necrotica
- Erosive pustular dermatosis

Secondary Cicatricial

- Traumatic injuries
- Traction alopecia
- Trichotillomania
- Pressure alopecia

Clinical approach to alopecia



CLINICAL APPROACH TO SCALP ALOPECIA

Majority of scalp (diffuse)

Common:
Telogen effluvium
Anagen effluvium

Less common:
Lichen planopilaris, diffuse
Alopecia areata, diffuse
Female pattern hair loss

Patterned

Widened part line(s)

Female pattern hair loss (Christmas tree pattern)
Traction alopecia

Frontal / Temporal

Common:
Male pattern hair loss (bitemporal)
Traction alopecia*
Less common:
Frontal fibrosing alopecia
Hyperandrogenism in women

Vertex / Midline

Male pattern hair loss
Female pattern hair loss
Central centrifugal cicatricial alopecia (primarily women of African descent)

Posterolateral

Alopecia areata (ophiasis)

Occipital

Pressure (postoperative) alopecia

Circumscribed and non-scarring

Alopecia areata (limited patch)
Tinea capitis*
Trichotillomania
Secondary syphilis
Pressure (postoperative) alopecia

Circumscribed and scarring

Inflammatory

Central centrifugal cicatricial alopecia
Lichen planopilaris (LPP)
Frontal fibrosing alopecia
Discoid lupus erythematosus (DLE)
Folliculitis decalvans
Dissecting cellulitis
Acne keloidalis

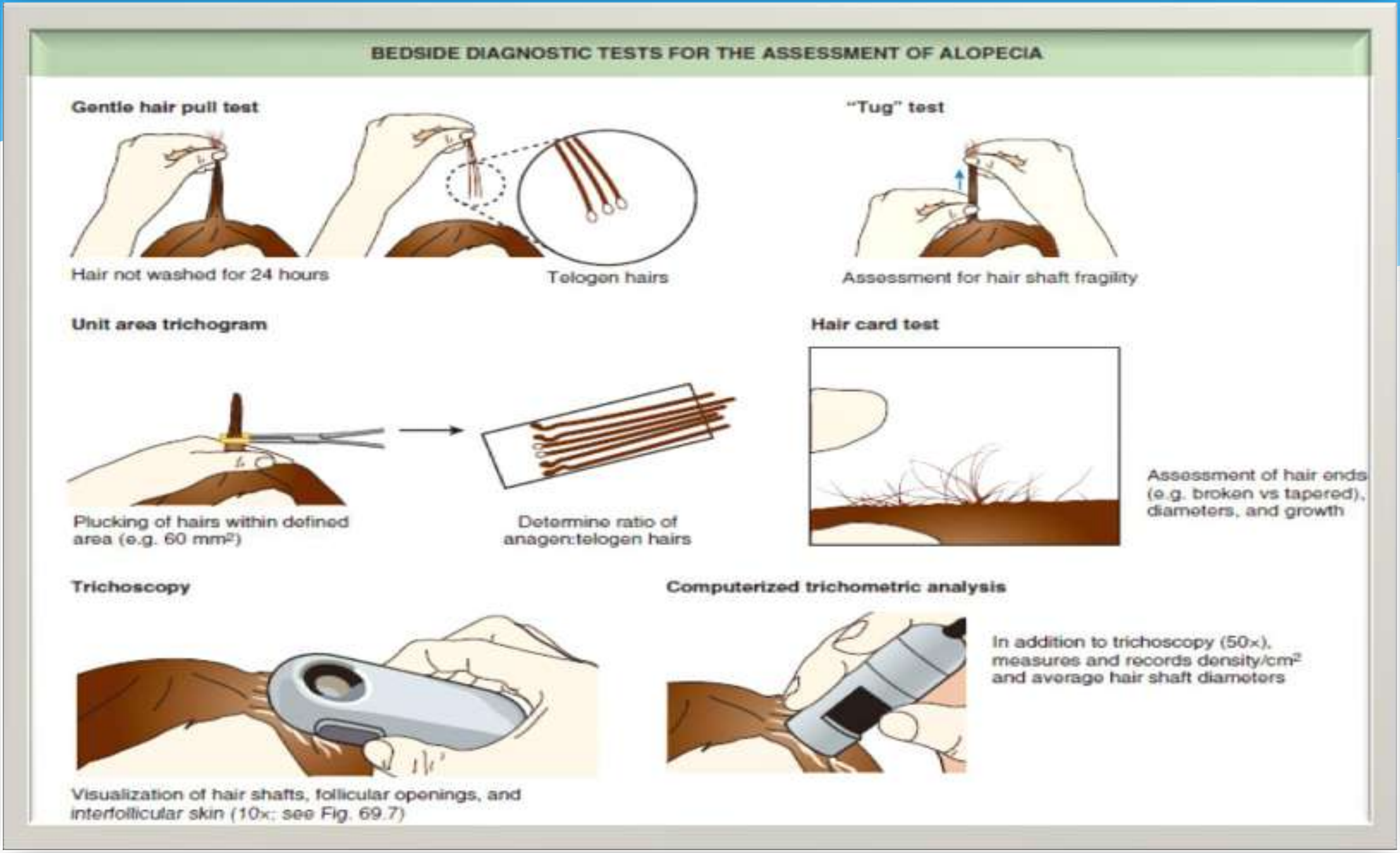
Non-inflammatory

Brocq's alopecia (non-inflammatory end-stage of LPP > DLE or folliculitis decalvans)

*if chronic or severe, can become scarring

diagnostic test for alopecia

Hair pull test: grasping 40–60 closely grouped hairs and applying gentle traction
Positive when more than 10% of hairs are easily pulled out.
Trichoscopy: examination of the hair follicle, hair shaft, and scalp with a dermoscope
Skin biopsy:



BEDSIDE DIAGNOSTIC TESTS FOR THE ASSESSMENT OF ALOPECIA

Gentle hair pull test
Hair not washed for 24 hours
Telogen hairs
"Tug" test
Assessment for hair shaft fragility
Unit area trichogram
Plucking of hairs within defined area (e.g. 60 mm²)
Determine ratio of anagen:telogen hairs
Hair card test
Assessment of hair ends (e.g. broken vs tapered), diameters, and growth
Trichoscopy
Visualization of hair shafts, follicular openings, and interfollicular skin (10×; see Fig. 69.7)
Computerized trichometric analysis
In addition to trichoscopy (50×), measures and records density/cm² and average hair shaft diameters

Alopecia, nonscarring

- Diffuse

1. telogen effluvium:

- Hair shedding, acute onset
- Reactive process response to physical event (surgery, high fever, thyroid disease, iron deficiency),
- medication, severe mental or emotional stress

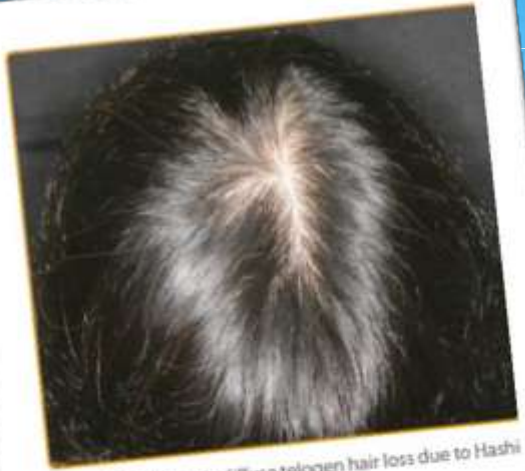


Figure 86-1 Chronic diffuse telogen hair loss due to Hashimoto disease.

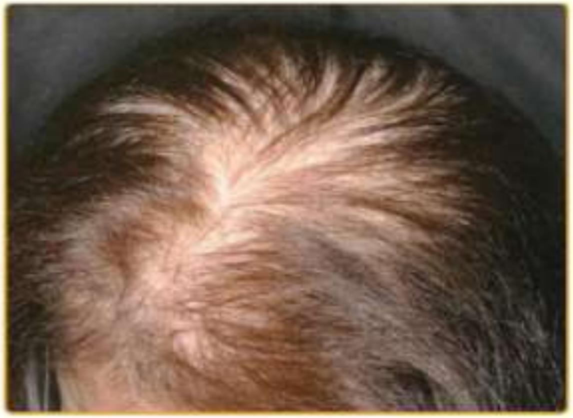
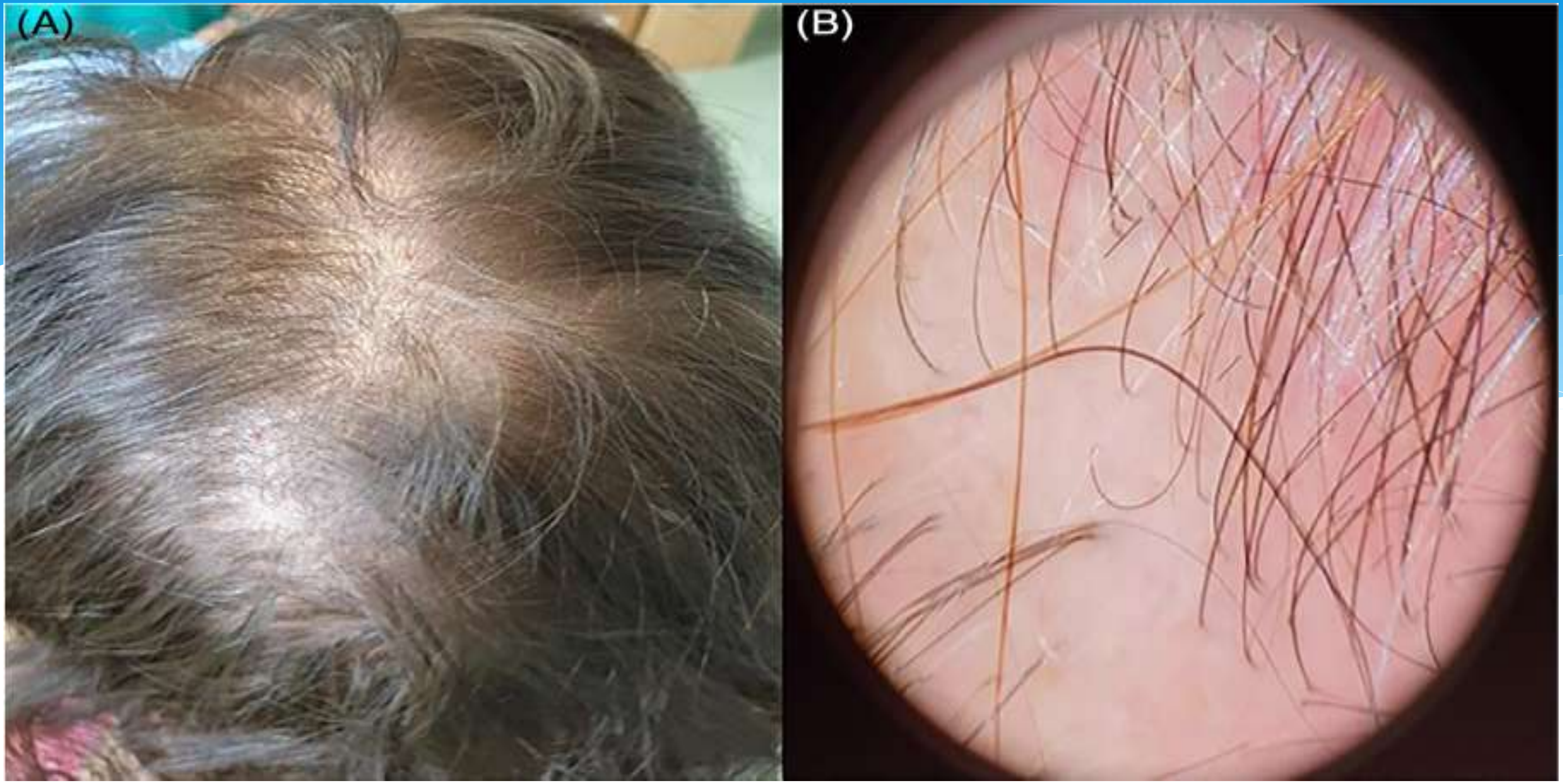


Figure 86-4 Acute telogen effluvium triggered by the administration of warfarin.



- Large number of hairs shift from anagen to telogen
- Positive pull test: more than 6 telogen hairs

Treatment

Telogen effluvium is self-correcting

- Recommendations include:
- Gentle handling of the hair, avoiding over-vigorous combing, brushing and any type of scalp massage
- Treat any underlying scalp disorder or hormonal problem determined, if any
- Ensure a nutritious diet, with plenty of protein, fruit and vegetables.
- Correct any abnormality in thyroid function, or levels of iron, vitamin B12 and folic acid.

Prognosis: 6 months if inciting cause is reversed Regrowing hairs with tapered or pointed hairs can be seen in the recovery phase

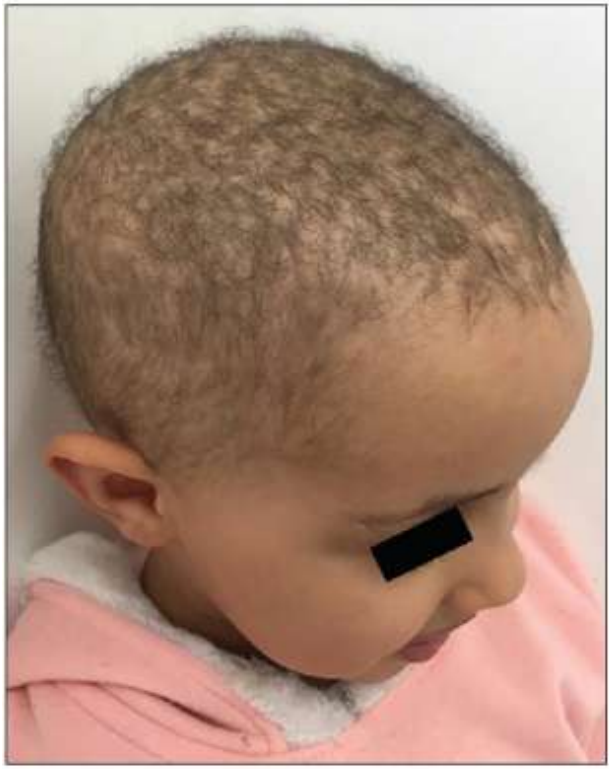
☐ Alopecia, nonscarring

➢ Diffuse

2. **Loose anagen syndrome**

- Fair-haired children with thin, sparse, hair; no need for haircuts; easily dislodgable hair
- Examination reveals sparse growth of thin, fine hair and diffuse or patchy alopecia

Figure 1: Short and spars hair in a 5-year-old girl



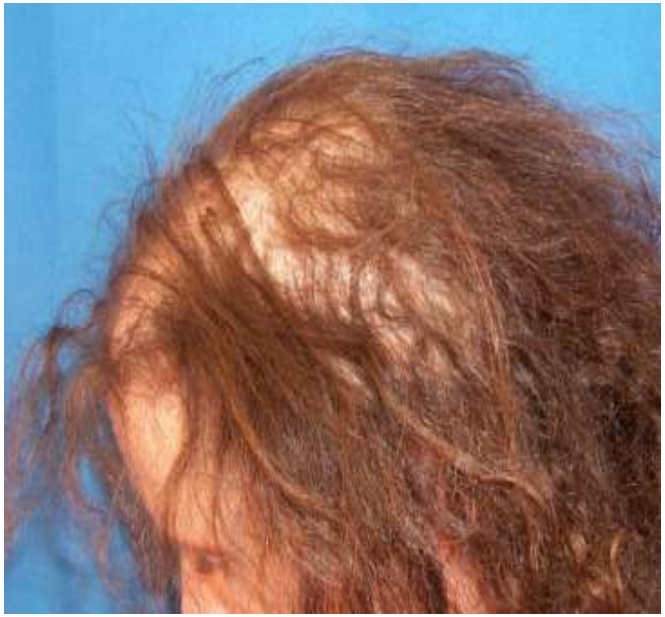
[redacted]

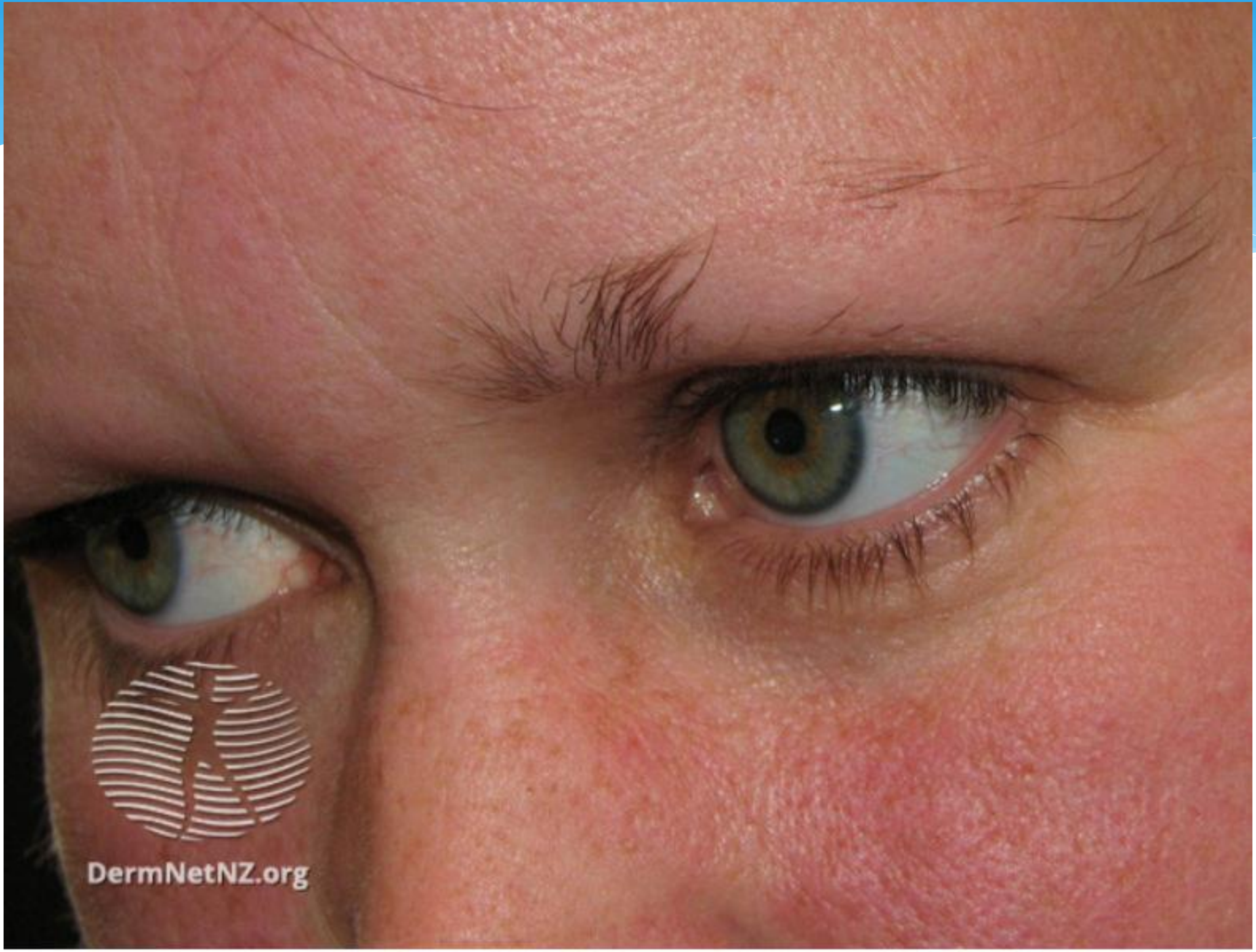
- Anagen hairs are easily and painlessly pulled from scalp
- **Diagnosis:** epilated hairs are predominantly in anagen phase;
- Hair mount shows distorted anagen bulb, ruffled cuticle
- **Improves with age**

- ☐ **Alopecia, nonscarring**
  - Diffuse

3. **Anagen effluvium (aka anagen arrest)**

- Hair broken off and not shed
- Radiation therapy and chemotherapy agents; occurs 2 to 4 weeks after treatment
- Hair shafts are abruptly thinned and break off at skin surface

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- Other causes: mercury intoxication, boric acid intoxication, thallium poisoning, colchicine, severe protein deficiency

Treatment

Anagen effluvium due to chemotherapy is expected to recover fully within 3–6 months of stopping it. The hair nearly always grows back normally, but sometimes patients with straight hair develop curly hair when it regrows. Hair color may also change.

Treatments for anagen effluvium include:

- Topical minoxidil solution
- Scalp cooling during chemotherapy
- Cosmetic camouflage to eyebrows

Alopecia, nonscarring

Patchy

1. **Alopecia areata**

- Abrupt onset
- Exclamation point hairs which are broken hairs that are tapered at the scalp
- Pigmented hair affected first, subsequently gray hair may also be targeted

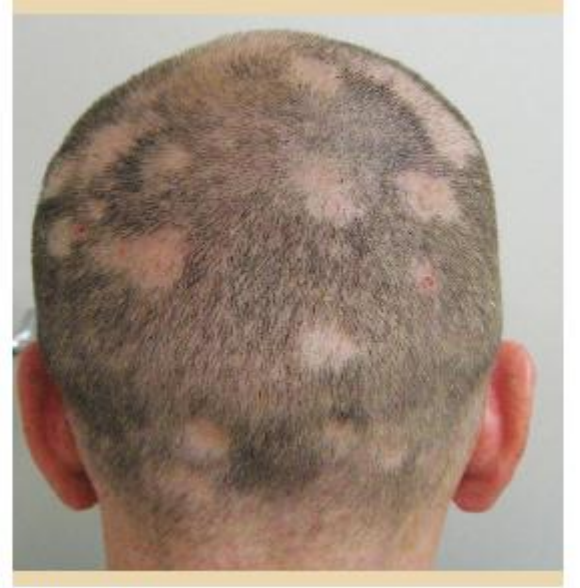

FIGURE 1-5 Patchy alopecia areata. *(Used with permission from Dr. Paradi Mirmirani.)*

Hair pull test positive for telogen hairs when disease is active

Follicular damage in anagen; then rapid transformation into telogen

- **Alopecia totalis:** total scalp hair loss
- **Alopecia universalis:** total scalp and body hair loss
- **Ophiasis:** localized hair loss along the periphery of the scalp
- **Nails:** pitting, mottled lunula,trachyonychia, or onychomadesis

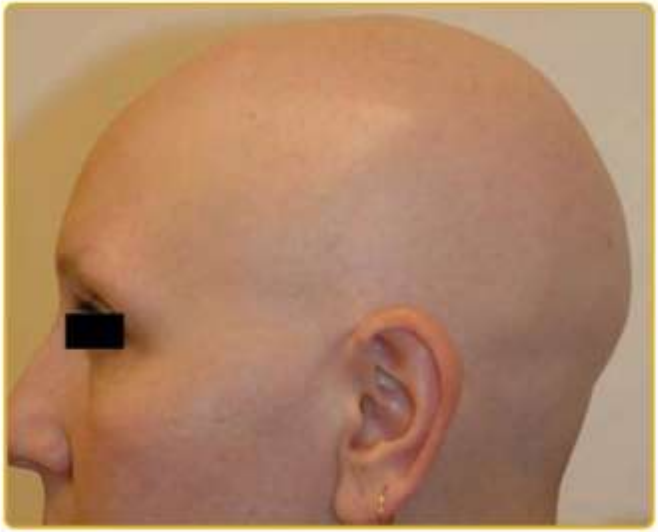
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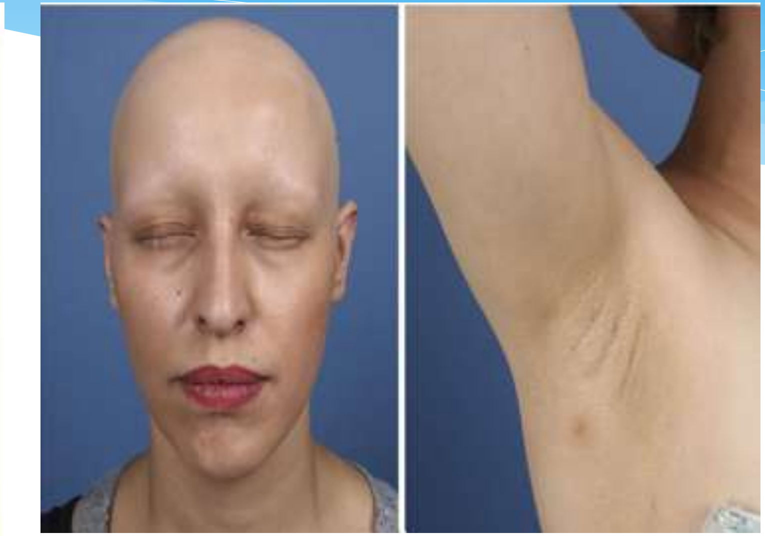
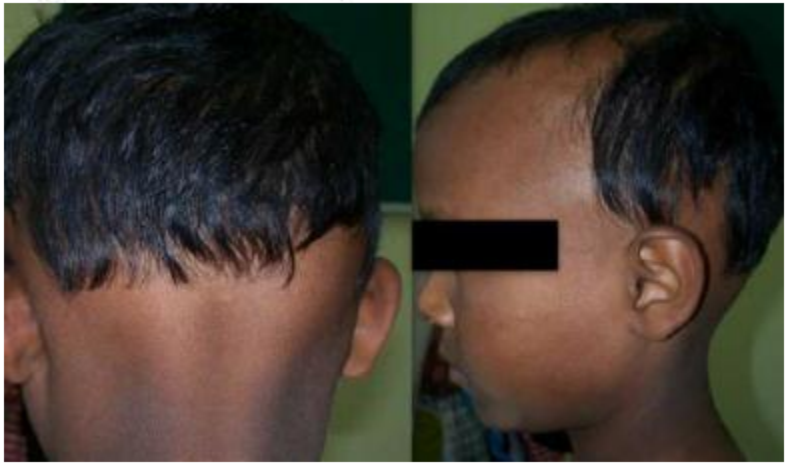
Figure 87-3 Patient with alopecia areata totalis.

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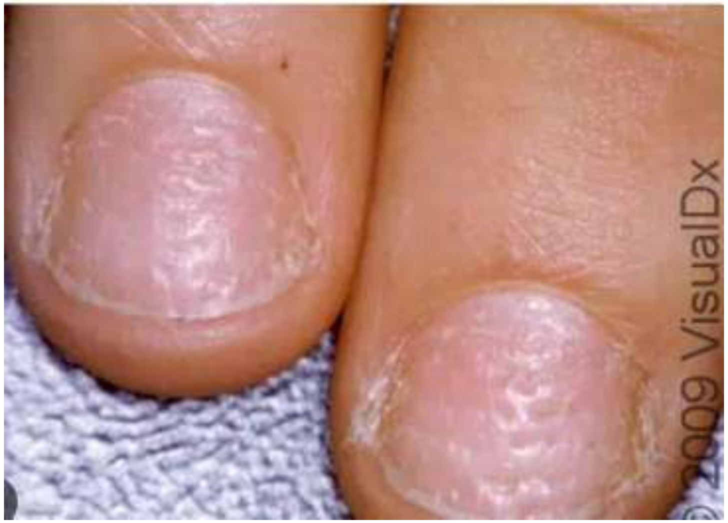
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- **Histology**: peribulbar infiltrate of T cells and macrophages (“swarm of bees”)
- **Associations:** In the patient: AD, thyroid

disease, vitiligo. In the family: atopic disorders, thyroid disease, vitiligo, diabetes mellitus, pernicious anemia, SLE (other autoimmune conditions)

Treatment

- **Patchy, or less than 50%:**
- Intralesional corticosteroid injections
- Topical treatments

  Potent corticosteroid

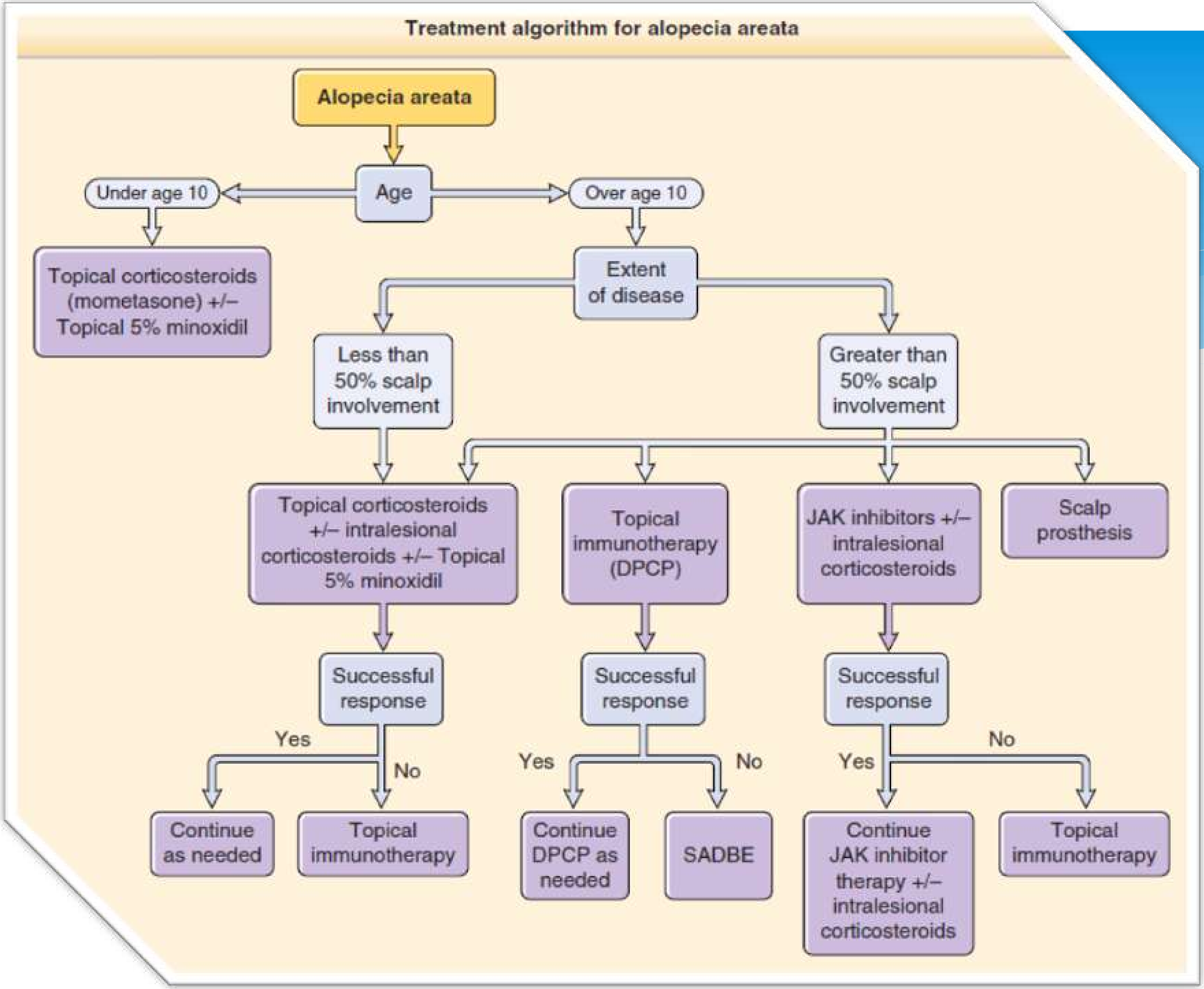
  Minoxidil solution

  Anthralin (dithranol) cream or ointment

- greater than 50% scalp involvement

- topical immunotherapy (squaric acid dibutylester [SADBE] or diphenylcyclopropenone [DPCP])
- systemic cortisone
- Janus kinase (JAK) inhibitors

In June 2022, The FDA approved baricitinib use in severe alopecia areata.



Alopecia, nonscarring

Patchy

2. **Trichotillomania**

- Impulse-control disorder
- Repeated plucking or pulling of hairs
- Confluence of short, sparse hairs within an otherwise normal area of the scalp
- Varying lengths of regrowth, “friar tuck” distribution of hair loss

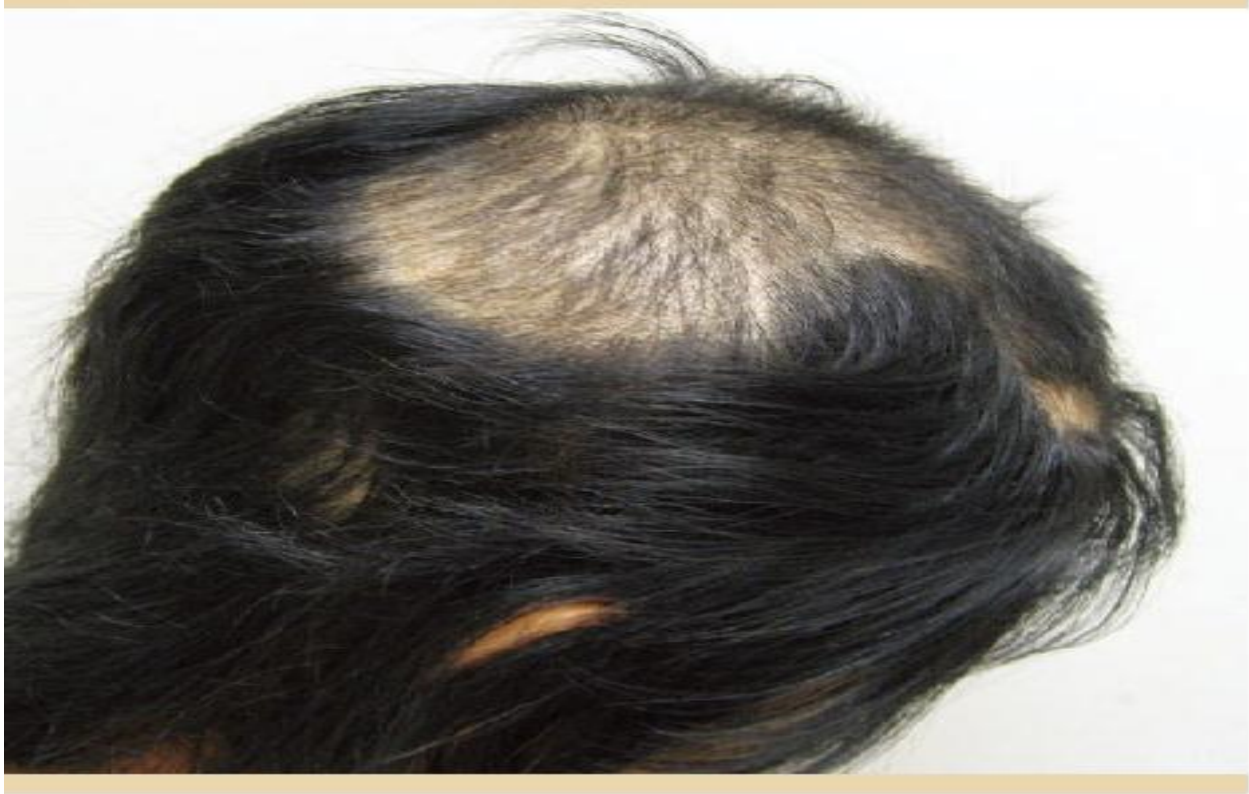


FIGURE 1-8 Trichotillomania. (*Used with permission*

- Regrowing hair is blunt tipped instead of pointed
- Eyebrows and upper eyelashes may be affected
- Often have other habits: nail biting, skin picking
- **Treatment:** psychological intervention and/or psychiatric medication to modify behavior

Alopecia, nonscarring

- Patchy

3. **Traction alopecia**

- Prolonged traction on the scalp by physical pressure: tight braids, foam rollers, tight pony tail, hair extensions

- The “fringe sign” is characteristic of marginal traction alopecia: the hair along the frontal hairline is retained but is attenuated or finer in caliber. The area of alopecia
is behind this “fringe”

Hair loss may be persistent if the traction is unrelenting

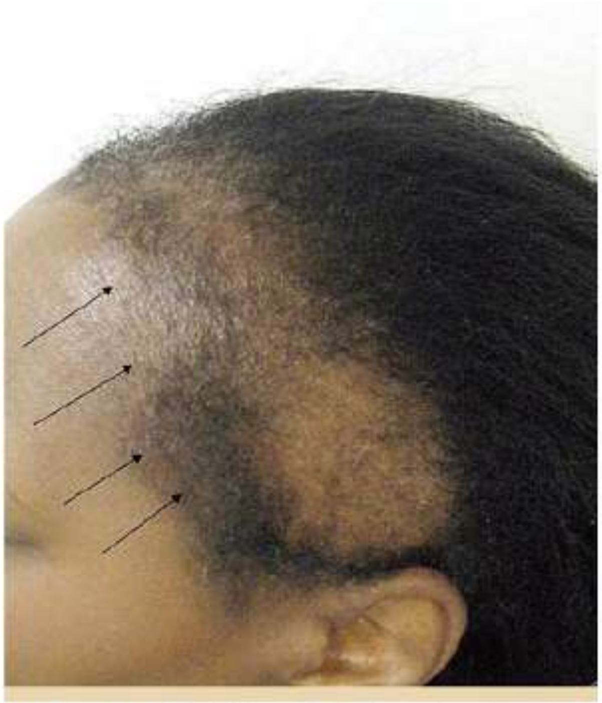


FIGURE 1-10 The “fringe sign” of traction alopecia involving the marginal hairline. (*Used with permission from Dr. Paradi Mirmirani.*)

Alopecia, nonscarring

Patchy

4. Triangular (temporal) alopecia.

- Triangular patch of vellus hairs or complete hair loss—either congenital or appears early in life
- Frontotemporal region
- Histology: vellus hairs
- No treatment, usually persistent

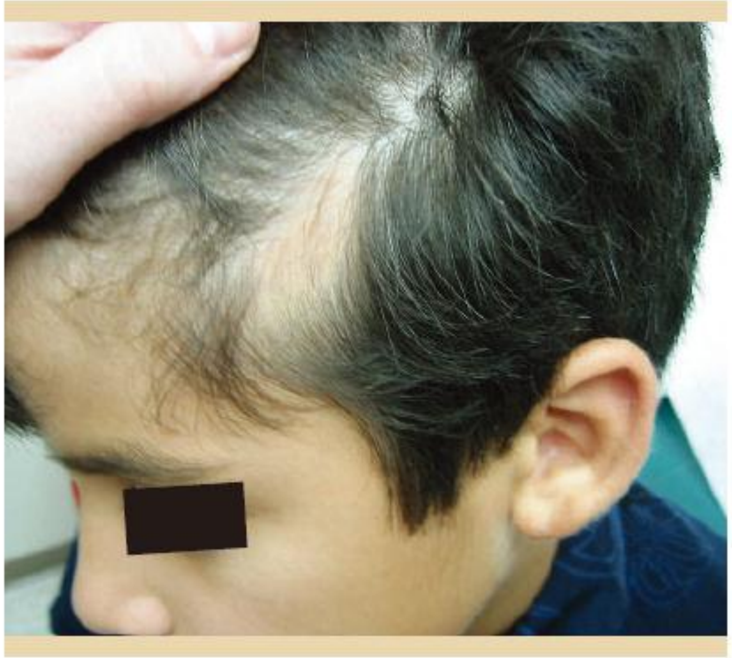


FIGURE 1-11 Triangular alopecia. (*Used with permission from Dr. Adelaide Hebert.*)

☐ Alopecia, nonscarring

- Patterned

5. **Androgenetic alopecia,**

- Hereditary thinning in genetically susceptible men and women
- Circulating testosterone (T) is converted to dihydrotestosterone (DHT) by 5-a-reductase enzyme at the target tissue.
- DHT is the active androgen causing miniaturization of hairs in androgen-sensitive areas of scalp.

☐ **Alopecia, nonscarring**

➢ Patterned

- In women there may be some additional nonandrogen signals that lead to thinning, especially during menopausal years
- Anagen is shorter; number of follicles remains the same.

Paradoxically DHT enlarges hair in androgensensitive areas (beard, chest)

- **Male pattern:** potential areas of hair loss are the frontal, temporal, and vertex regions.

Hamilton-Norwood classification of male pattern hair loss

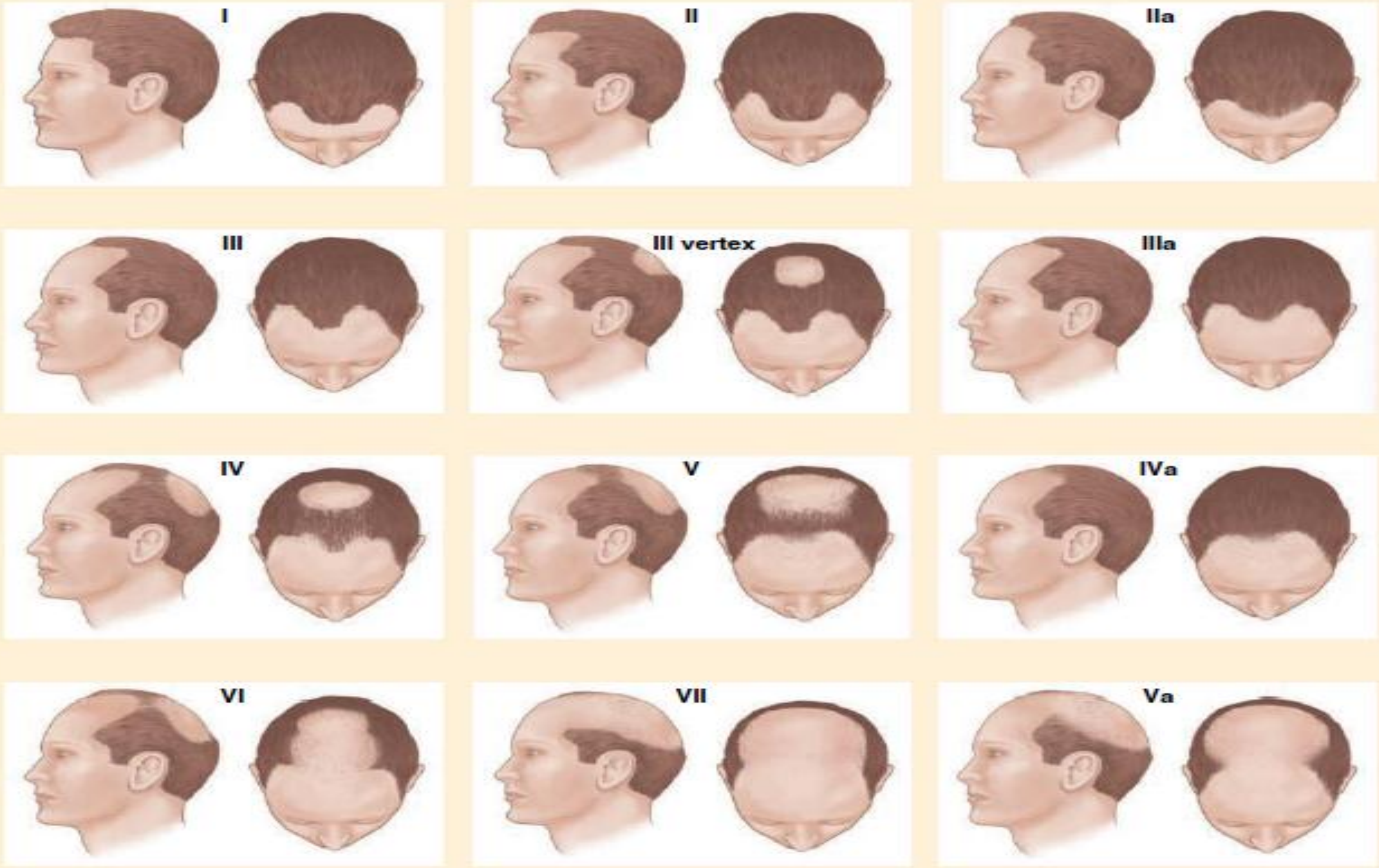

I
II
IIa
III
III vertex
IIIa
IV
V
IVa
VI
VII
Va

Figure 85-1 Hamilton-Norwood classification. (Redrawn from Blume-Peytavi U, Blumeyer A, Tosti A, et al. S1 guideline for diagnostic evaluation in androgenetic alopecia in men, women and adolescents. *Br J Dermatol*. 2011;164[1]:5-15.)

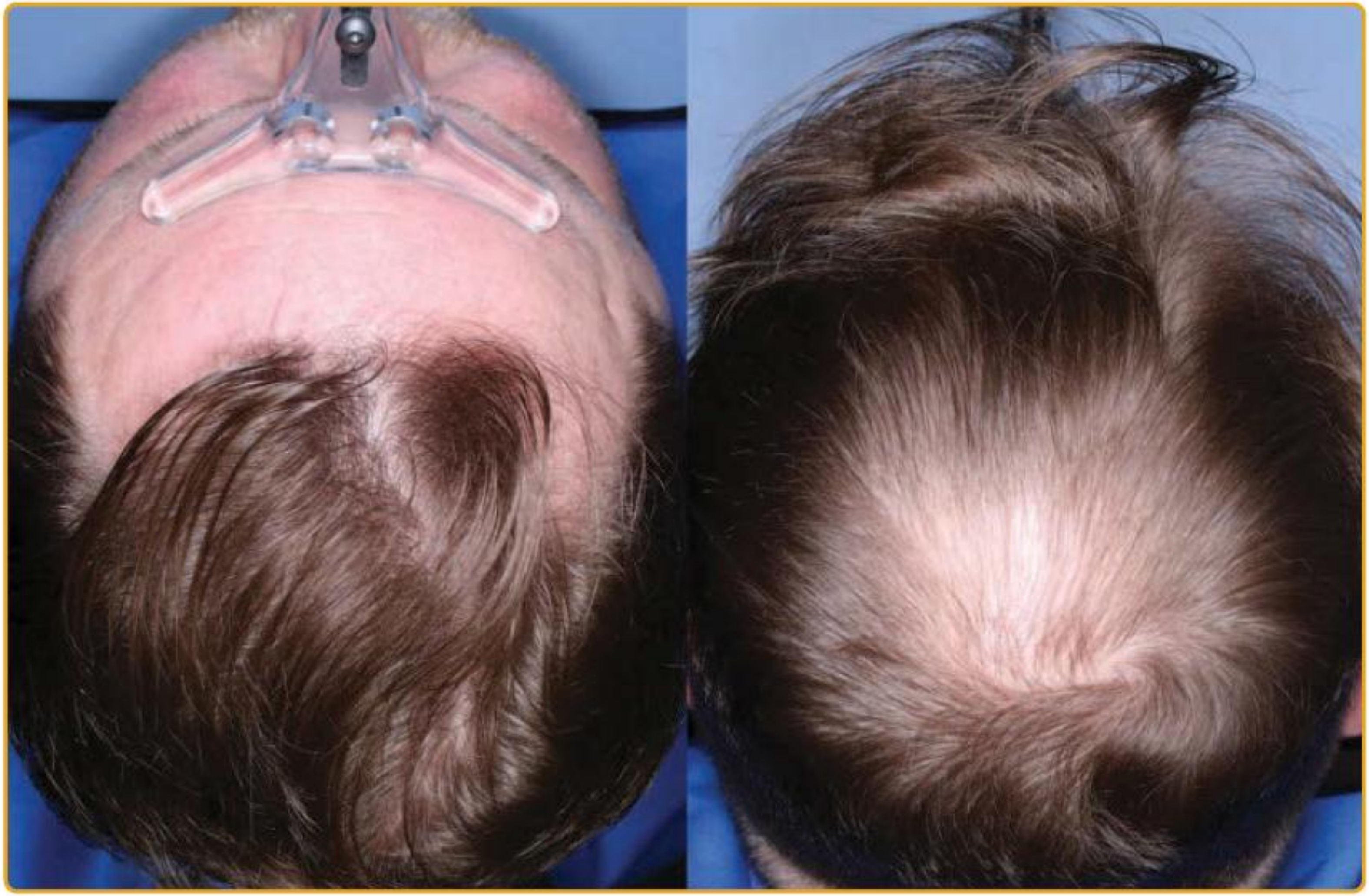
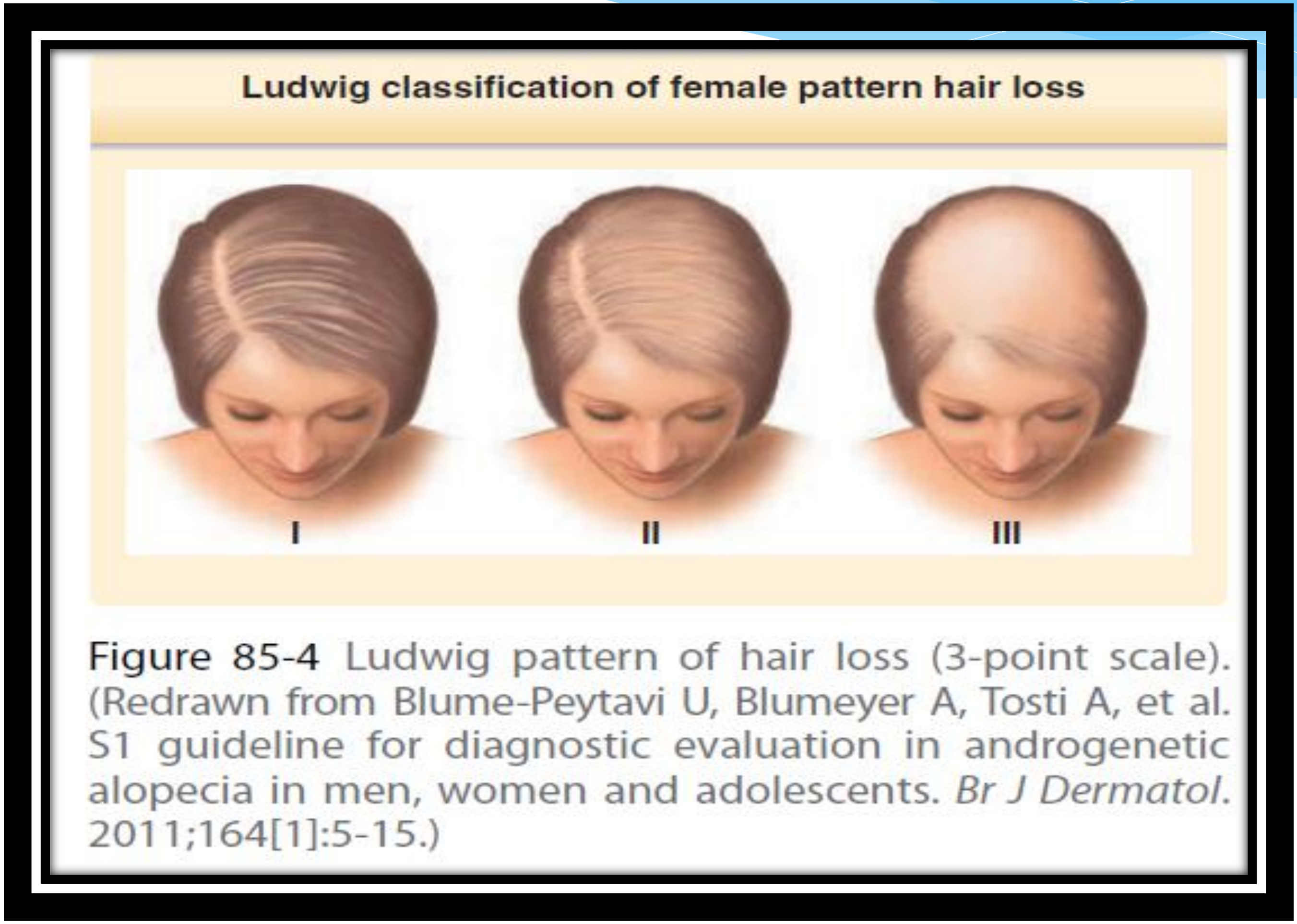


Figure 85-2 Male-pattern androgenetic alopecia, grade III vertex according to the Hamilton-Norwood classificati
Standardized global photography overview (Canfield system).

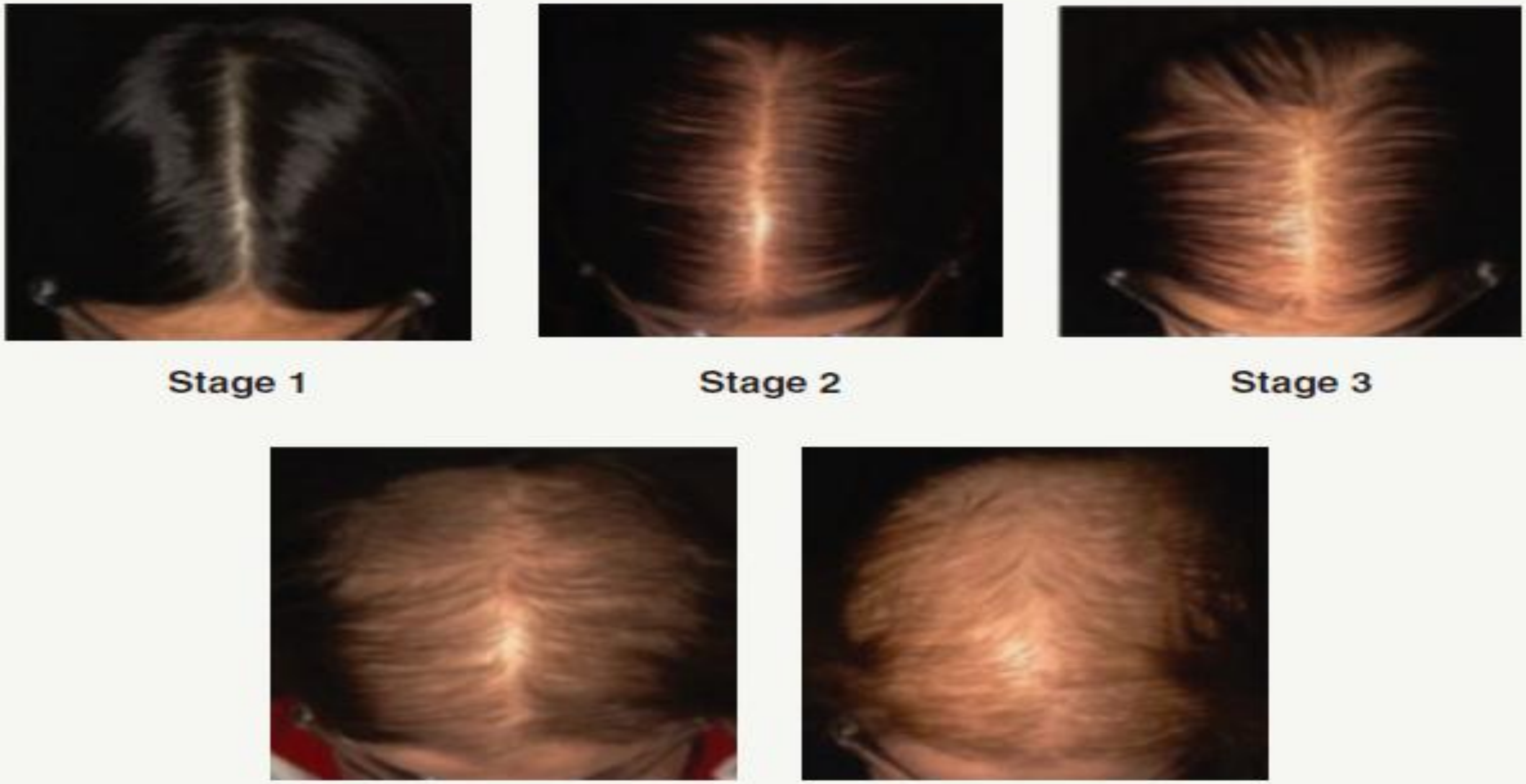
- **Female pattern:** diffuse thinning in the midscalp, vertex, and temporal areas; frontal hairline is retained (Ludwig classification)
- **Histology:** miniaturization increased vellus-to-terminal hair ratio, preserved sebaceous glands



Ludwig classification of female pattern hair loss

Figure 85-4 Ludwig pattern of hair loss (3-point scale). (Redrawn from Blume-Peytavi U, Blumeyer A, Tosti A, et al. S1 guideline for diagnostic evaluation in androgenetic alopecia in men, women and adolescents. *Br J Dermatol*. 2011;164[1]:5-15.)

THE SINCLAIR SCALE FOR FEMALE PATTERN HAIR LOSS



Stage 1

Stage 2

Stage 3

Stage 4

Stage 5

Fig. 69.5 The Sinclair scale for female pattern hair loss. Stage 1 – normal; Stage 2 – widening of the central part line; Stage 3 – widening of the part line with translucency of the hairs at its border; Stage 4 – development of a bald area anteriorly along the part line; and Stage 5 – advanced hair loss.

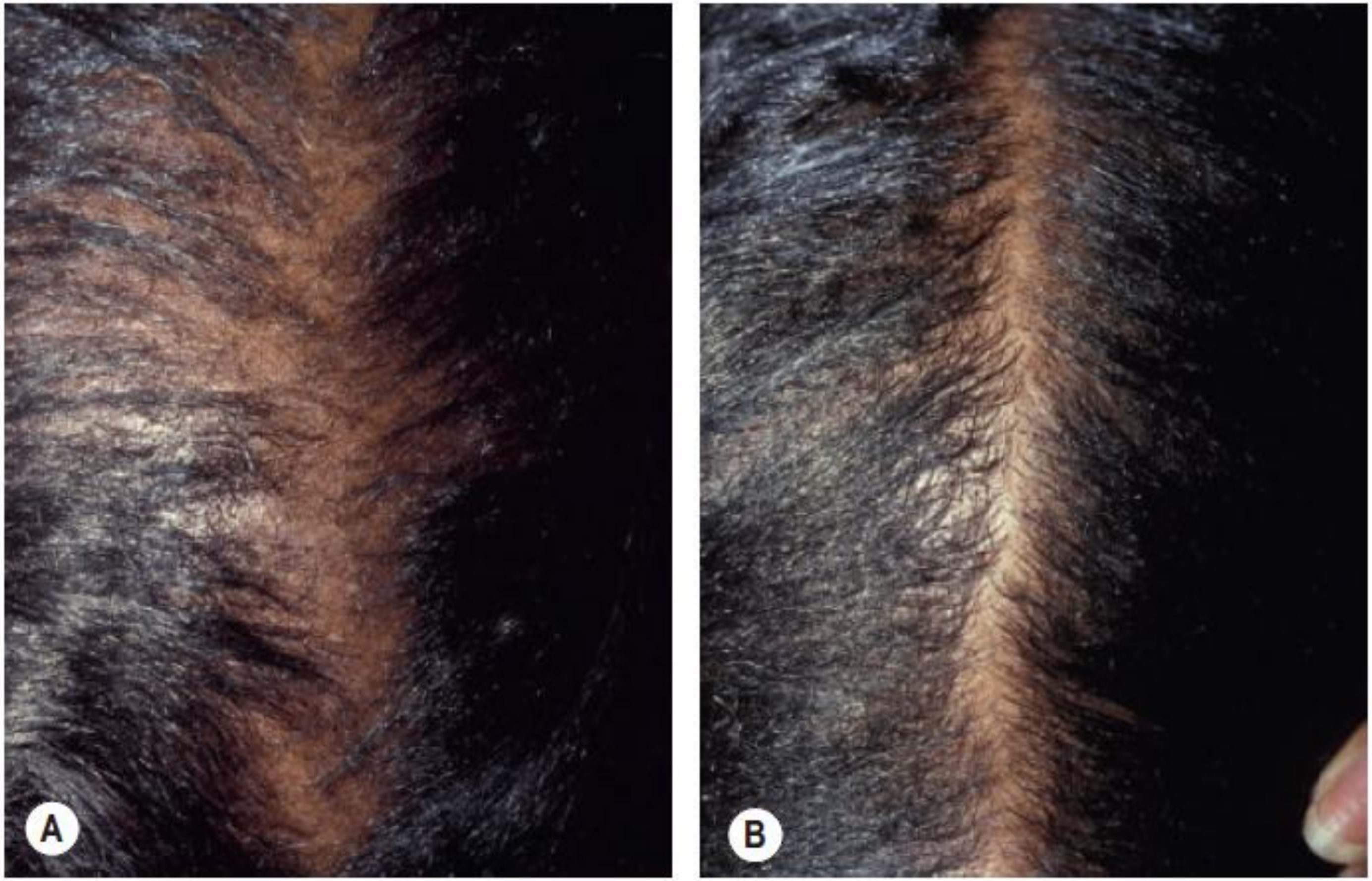


Fig. 69.6 Female androgenetic alopecia. Comparison of the part line on the crown (**A**) and the occiput (**B**).

Treatment

- Medical treatment:

–– Finasteride: 5-a-reductase type II inhibited

–– Minoxidil: increases the number of follicles in anagen, enlarges miniaturized hairs

- Surgical treatment: hair transplantation with minigrafts and micrografts

☐Alopecia, Scarring

Classification:

- **Primary:** hair follicle is the primary target of the immune attack, i.e., lichen planopilaris (LPP), folliculitis decalvans, and acne keloidalis
- **Secondary:** hair follicle is an innocent bystander which is nonspecifically affected by the inflammatory process, i.e., cutaneous sarcoidosis, burns, and chronic infections.

❑Alopecia, Scarring

1. **Pseudopelade of Brocq**

- Idiopathic lymphocytic primary cicatricial alopecia that predominantly affects the scalp.
- Women between 30 and 50 years of age are most frequently affected
- Oval- or irregularly shaped atrophic patches which may be mistaken for alopecia areata with patches of hair growth, “**footprints in the snow.**”
- No scalp redness or perifollicular scale

- **Histology:** atrophy, perifollicular inflammation at the level of the infundibulum, fibrosis that extends in to the subcutis

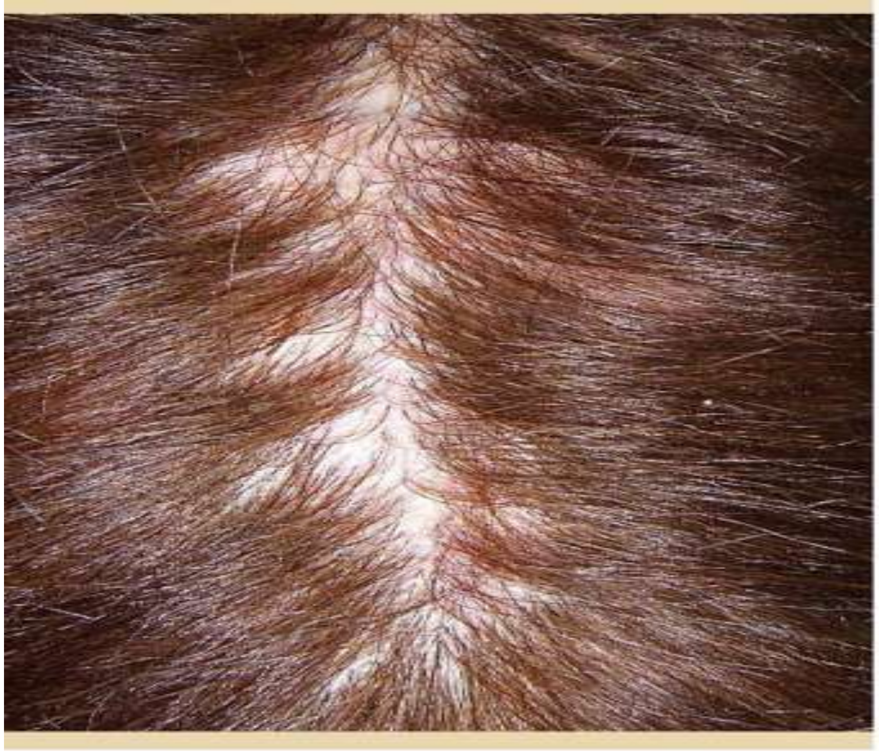
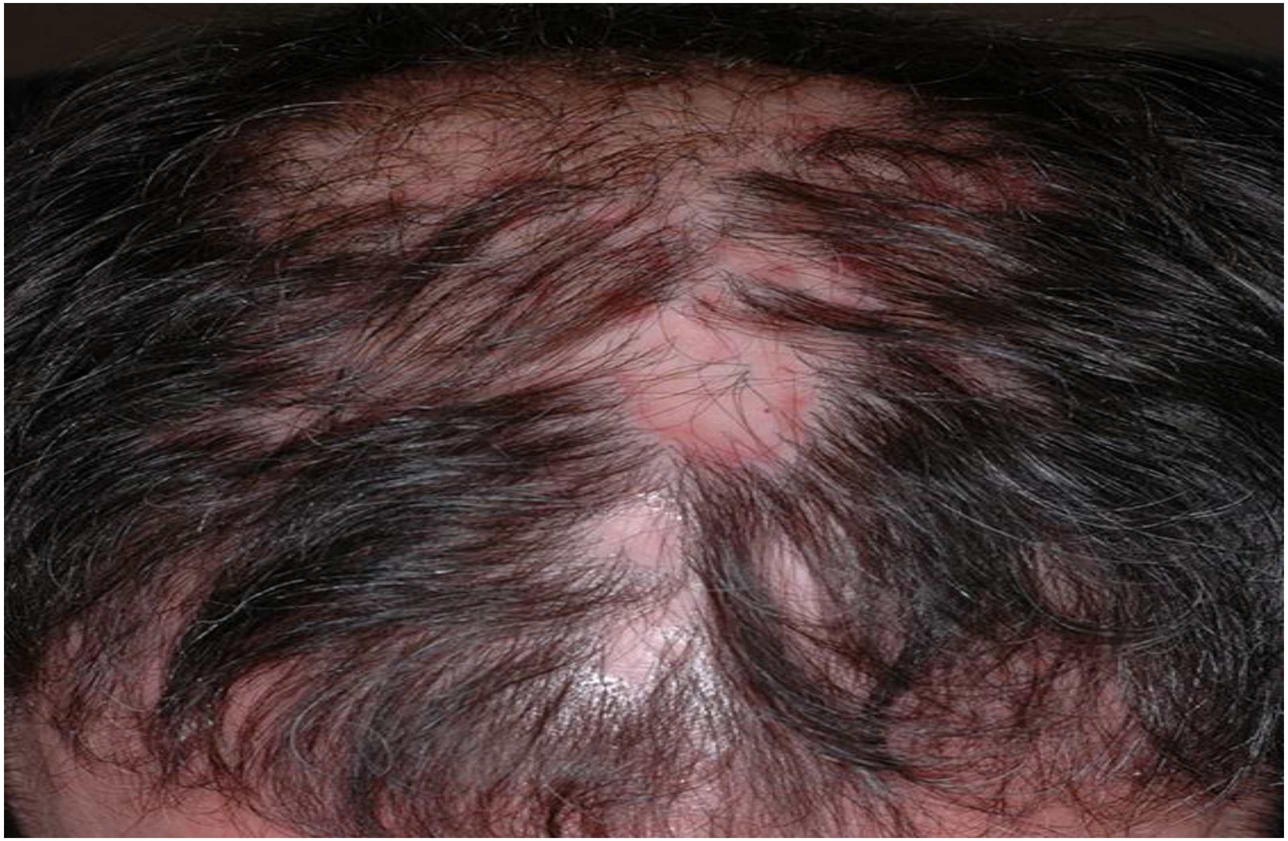


FIGURE 1-12 Pseudopelade (of Brocq).

☐Alopecia, Scarring

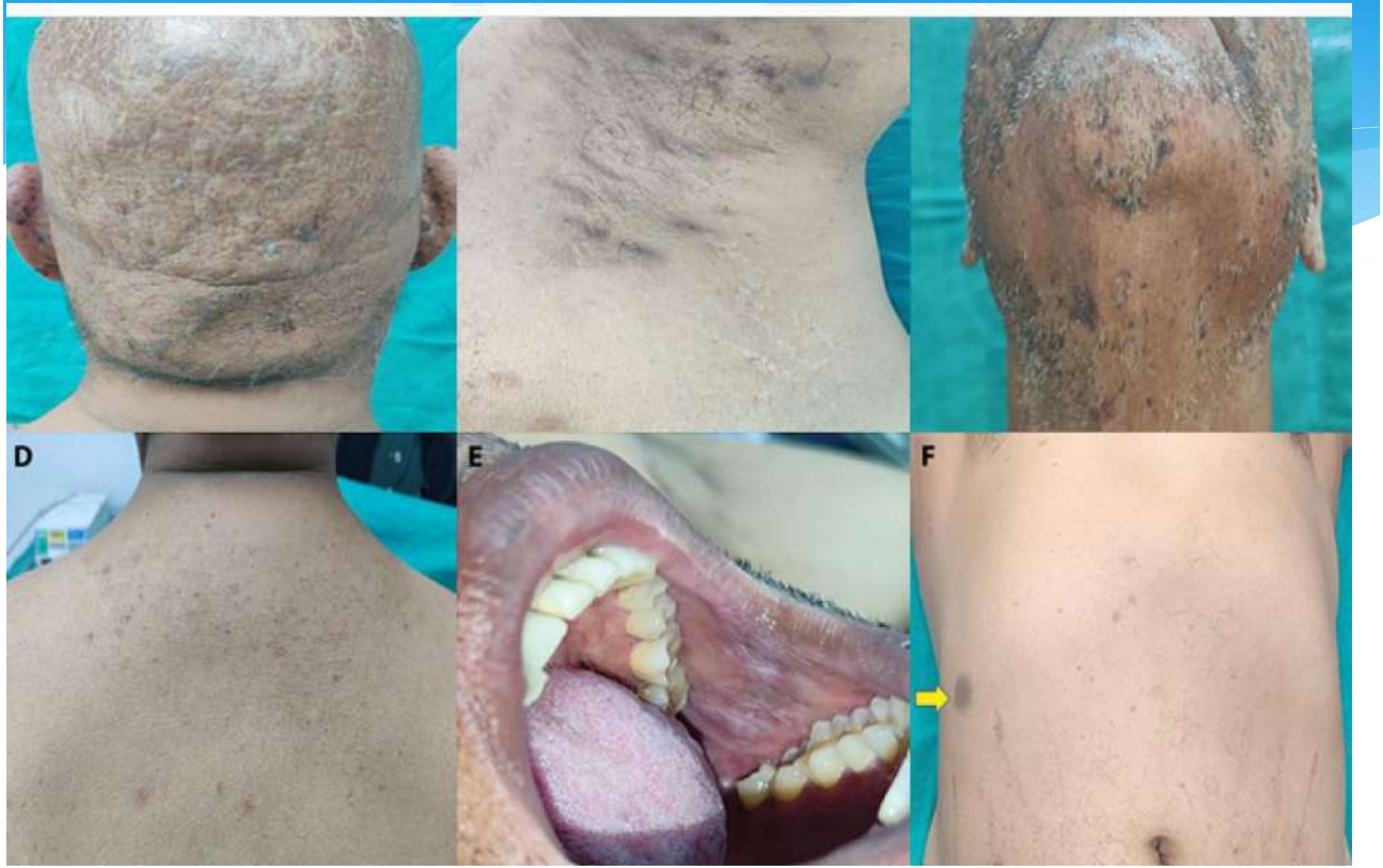
2. **Lichen planopilaris (LPP)**

- Perifollicular erythema and scale at the periphery of the patch of alopecia
- More than 50% associated with cutaneous or oral lichen planus
- Involves scalp alone or scalp and other hair-bearing areas ()



Graham Little syndrome

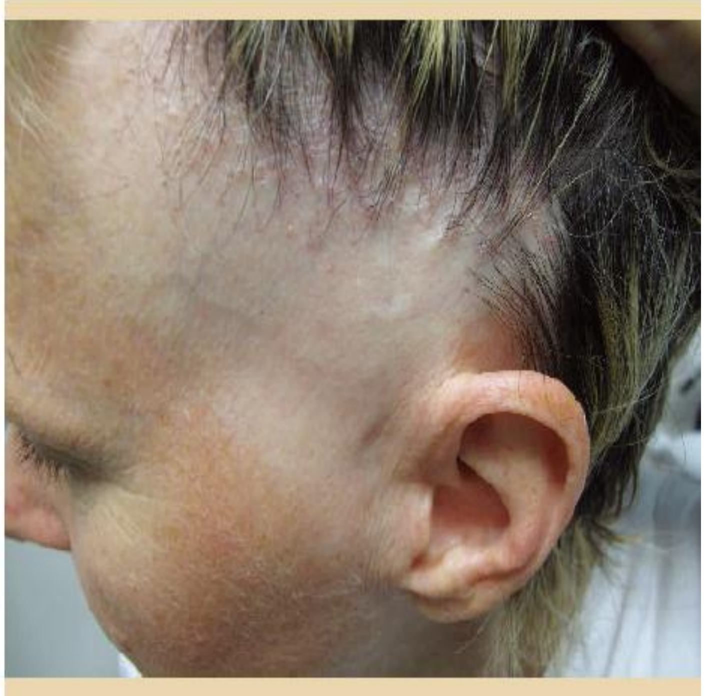
Patchy cicatricial alopecia of the scalp,
Non-cicatricial alopecia of the axilla and groin,
a follicular spinous papule on the body, scalp, or both

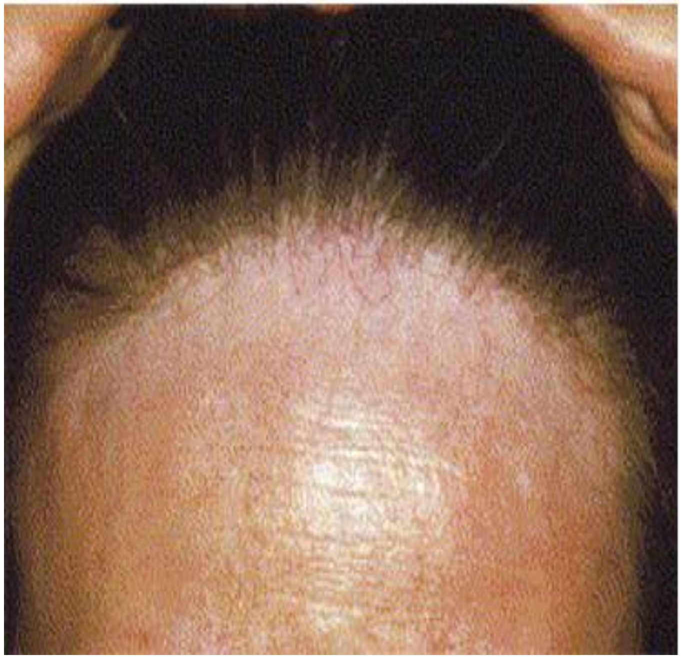

D
E
F

- **Histology:** typically same as LPP, may see lichenoid interface dermatitis of the superficial follicular epithelium

Alopecia，Scarring

3· **Frontal fibrosing alopecia:** frontotemporal hairline recession and eyebrow loss in postmenopausal women that is associated with perifollicular erythema and scaling, in a bandlike distribution along the frontotemporal hairline

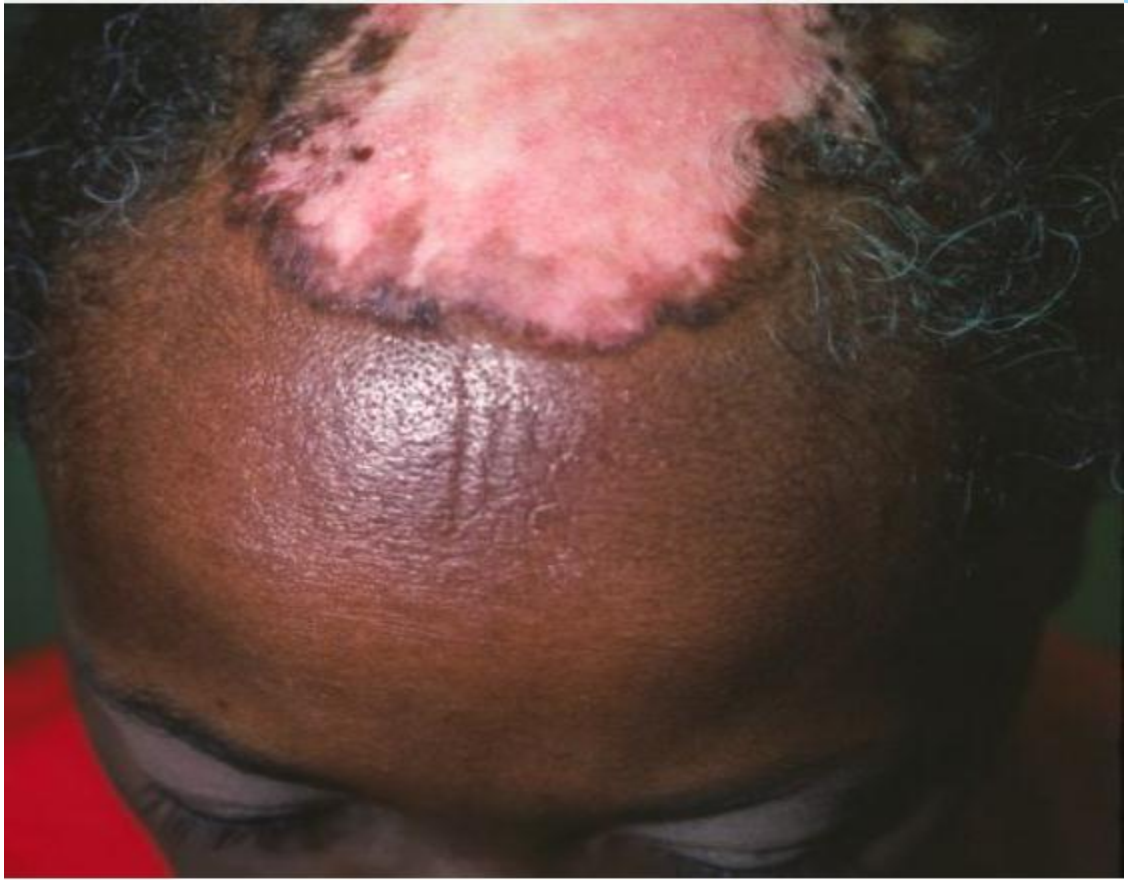




Alopecia，Scarring

4. Lupus erythematosus

- Chronic cutaneous (discoid) lupus erythematosus ： scarring alopecia, erythema, hypo- and hyperpigmentation of the scalp, dilated follicles plus keratin plugs, scaling at the center of the patch of alopecia

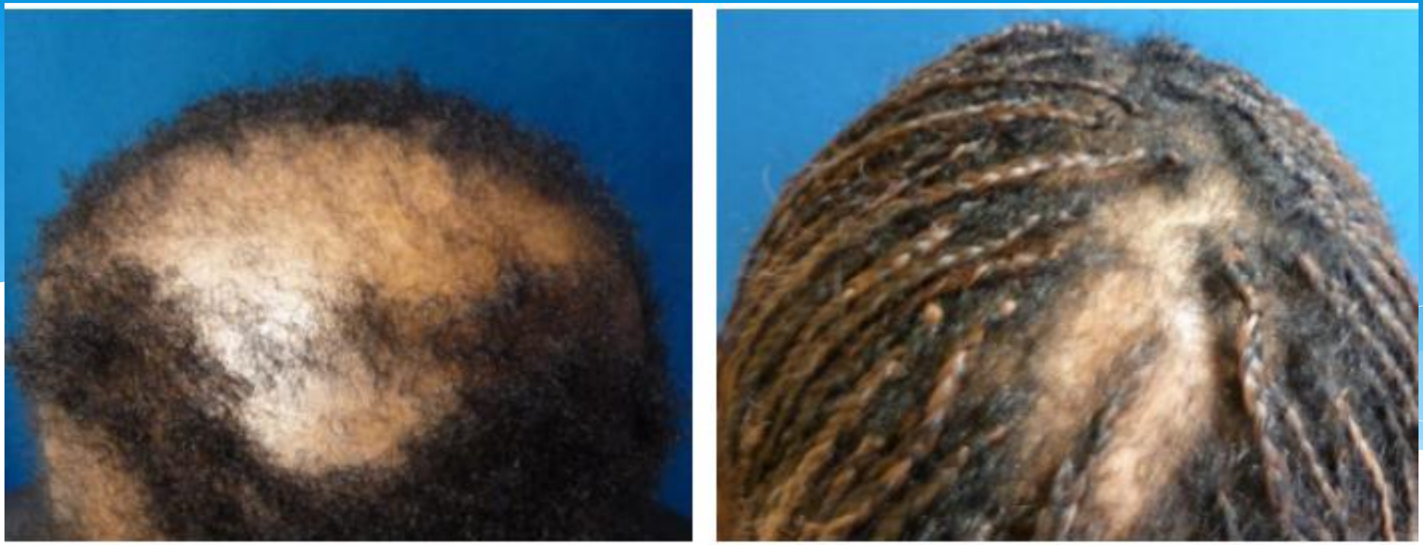


- **Systemic lupus erythematosus:** diffuse, nonscarring alopecia; broken hairs in frontal region (“lupus hairs”)
- Diagnostic biopsy and direct immunofluorescence
- **Treatment:** topical, intralesional, or oral steroids; systemic retinoids; antimalarials

❑Alopecia, Scarring

4. **Central centrifugal cicatricial alopecia (CCCA)**

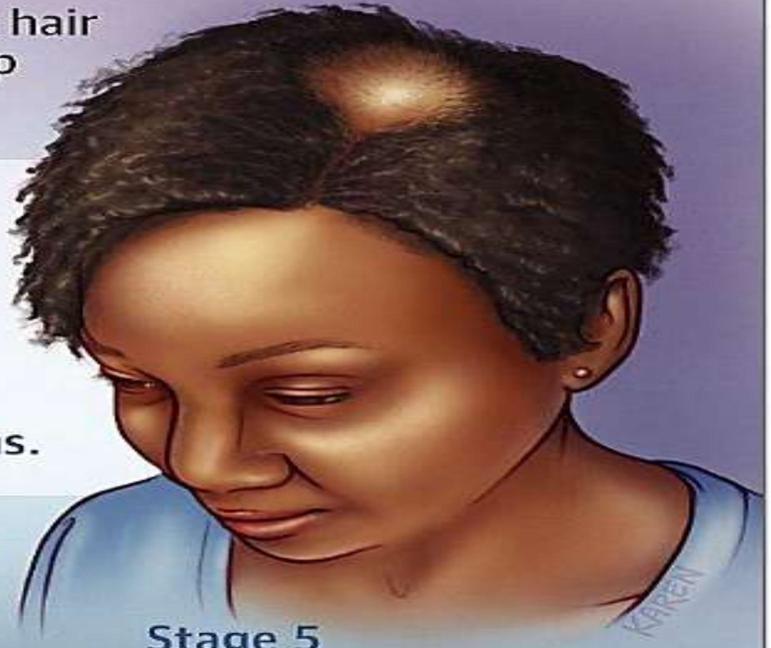
- Previously called follicular degeneration syndrome; hot-comb alopecia
- Follicular loss mainly on the crown of the scalp
- Possibly secondary to hair care practices



- **Histology:** premature desquamation of the inner root sheath, mononuclear infiltrate at the isthmus, loss of the follicular epithelium with fibrosis

Central centrifugal cicatricial alopecia (CCCA)

CCCA is a disease characterized by permanent hair loss that begins in the central crown of the scalp and widens over time.



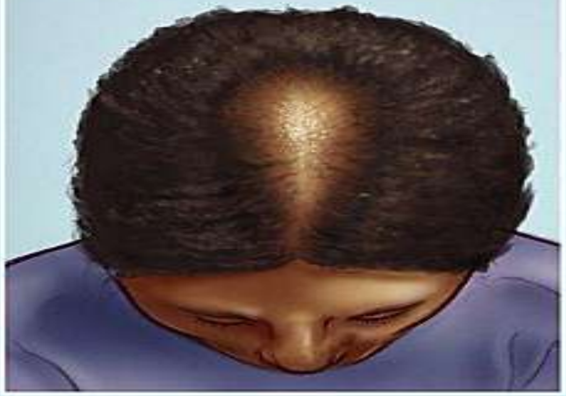
CCCA occurs almost exclusively in black women over the age of 30.

Symptoms include burning, itching, tenderness, redness, scaling or flaking, and acne-like bumps on the scalp.

Some people do not experience symptoms.

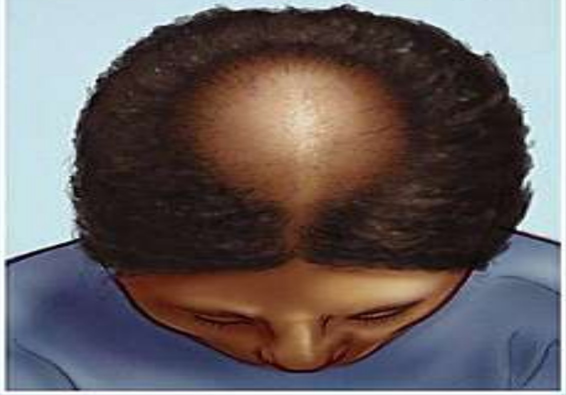
Typical progression of CCCA

Stage 1



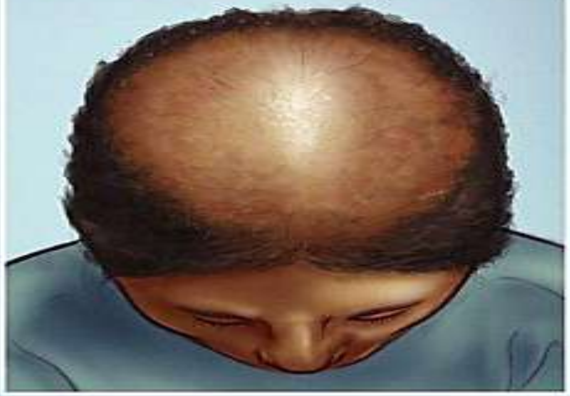
Subtle hair thinning in the central crown

Stage 3



Increased hair thinning with central scarring

Stage 5



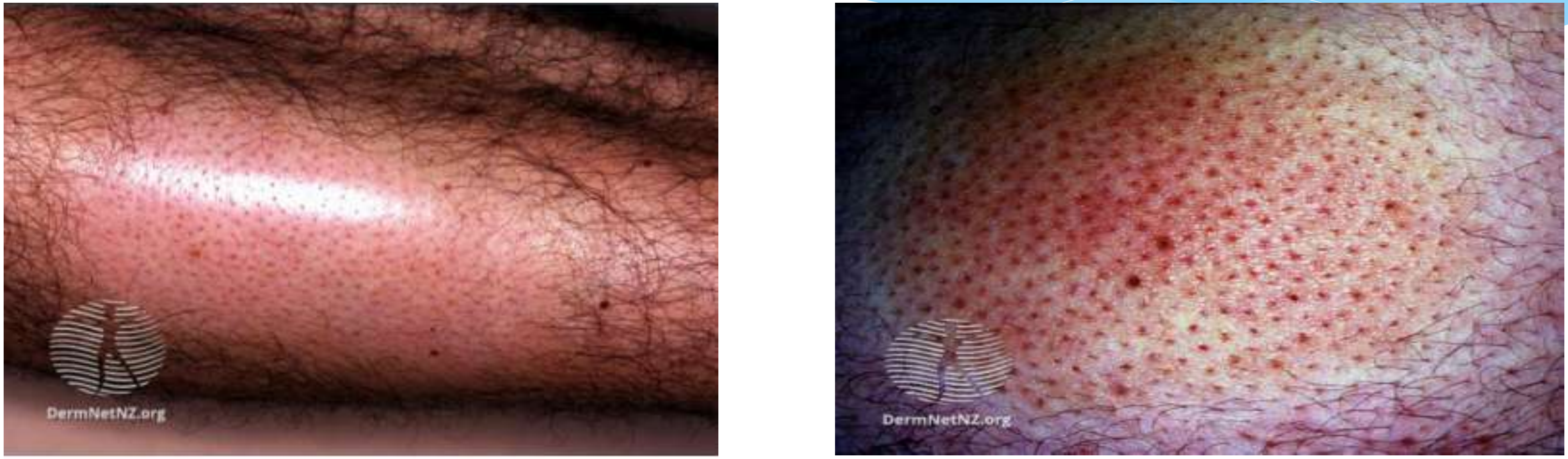
Severe, permanent hair loss and scarring

Treatment of CCCA is dependent on the stage of disease and focuses on relieving symptoms, slowing or stopping hair loss, and promoting hair regrowth in areas that are not permanently scarred.

❑ Alopecia, Scarring

5. **Alopecia mucinosa (follicular mucinosis)**

- Erythematous plaques or flat patches without hair
- Children: head and neck, benign, self-resolving
- Adults: more widespread distribution; may be associated with cutaneous T-cell lymphoma


DermNetNZ.org
DermNetNZ.org

- **Histology:** mucin in the outer root sheath and sebaceous glands, perifollicular lymphohistiocytic infiltrate

❑ Alopecia, Scarring

6.**Dissecting cellulitis:** Perifolliculitis capitis abscedens et suffodiens

- May be part of the follicular occlusion triad (cystic acne, hidradenitis, dissecting cellulitis)
- Fluctuant nodules on vertex, occiput, sterile pus
- **Histology:** sinus tracts, sterile abscesses
- **Treatment:** systemic steroids, systemic antibiotics, dapsone, retinoids, biologics, surgical excision

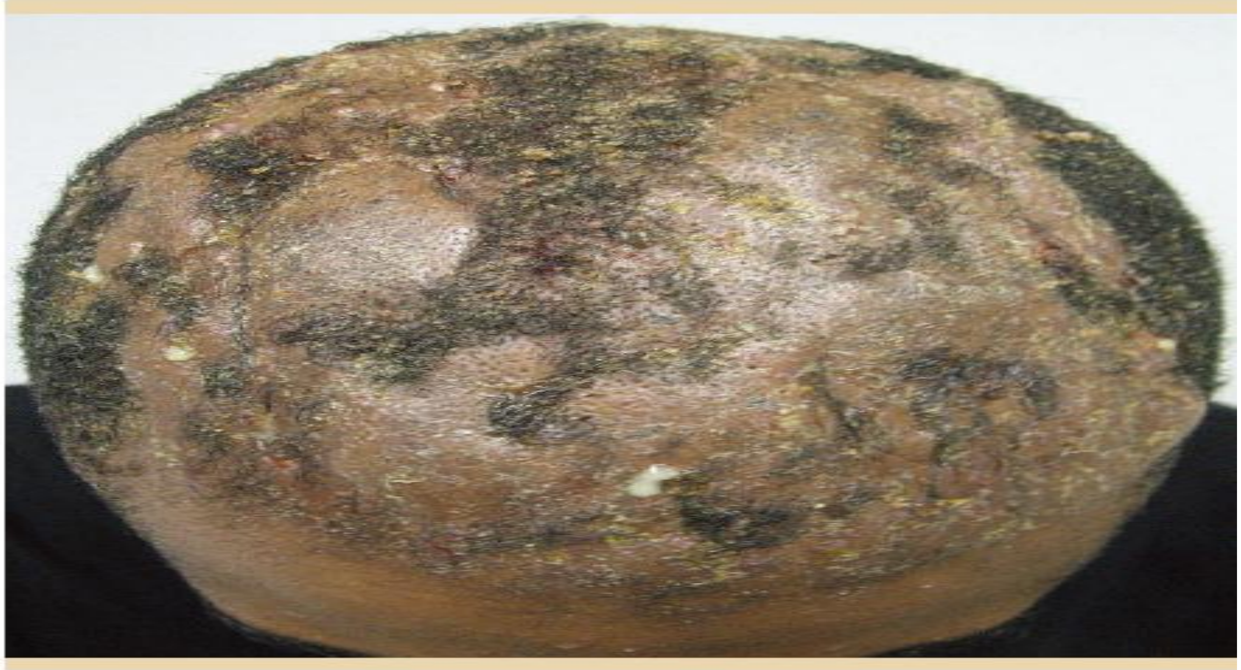


FIGURE 1-17 Dissecting cellulitis. (*Used with permission from Dr. Paradi Mirmirani.*)

❑Alopecia, Scarring

7. Folliculitis decalvans

- Scarring alopecia with crusting, pustules, and erosions
- Staphylococcus aureus usually cultured
- **Histology:** acute suppurative folliculits with neutrophils and eosinophils; later mixed with lymphocytes and histiocytes
- Loss of sebaceous epithelium and perifollicular fibrosis

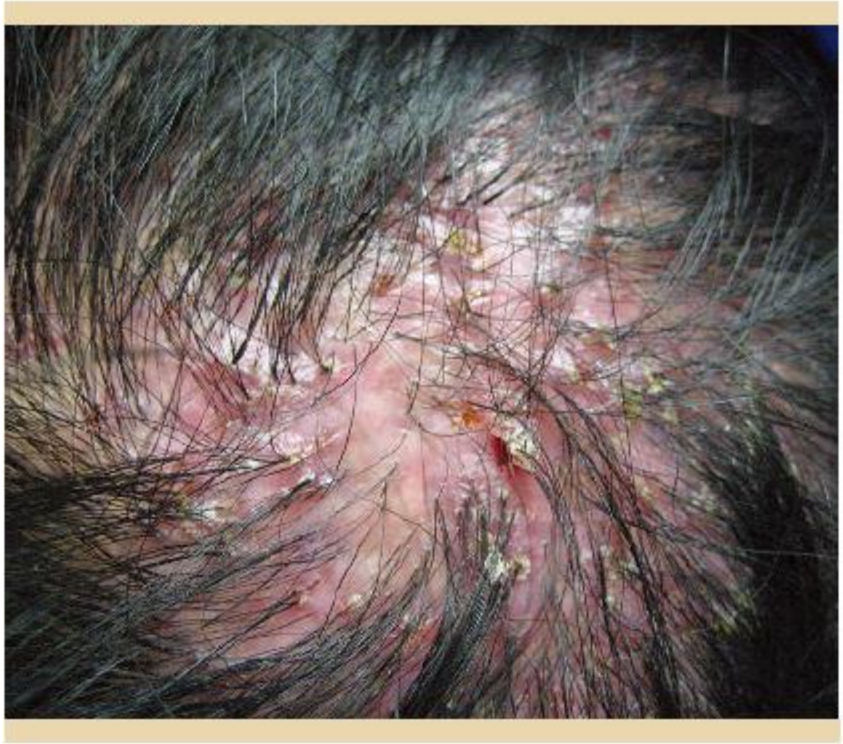


FIGURE 1-18 Folliculitis decalvans. (*Used with permission from Dr. Paradi Mirmirani*.)

- **Treatment:** staphylococcal eradication: systemic antibiotics with or without rifampin, systemic and/or topical steroids

☐ Alopecia, Scarring

8. **Acne keloidalis**

- Follicular pustules and papules that progress to firm, keloidal papules
- Commonly on occiput of patients with coarse and/or curly hair
- Foreign-body reaction to trapped hair shaft fragments
- Often bacterial superinfection

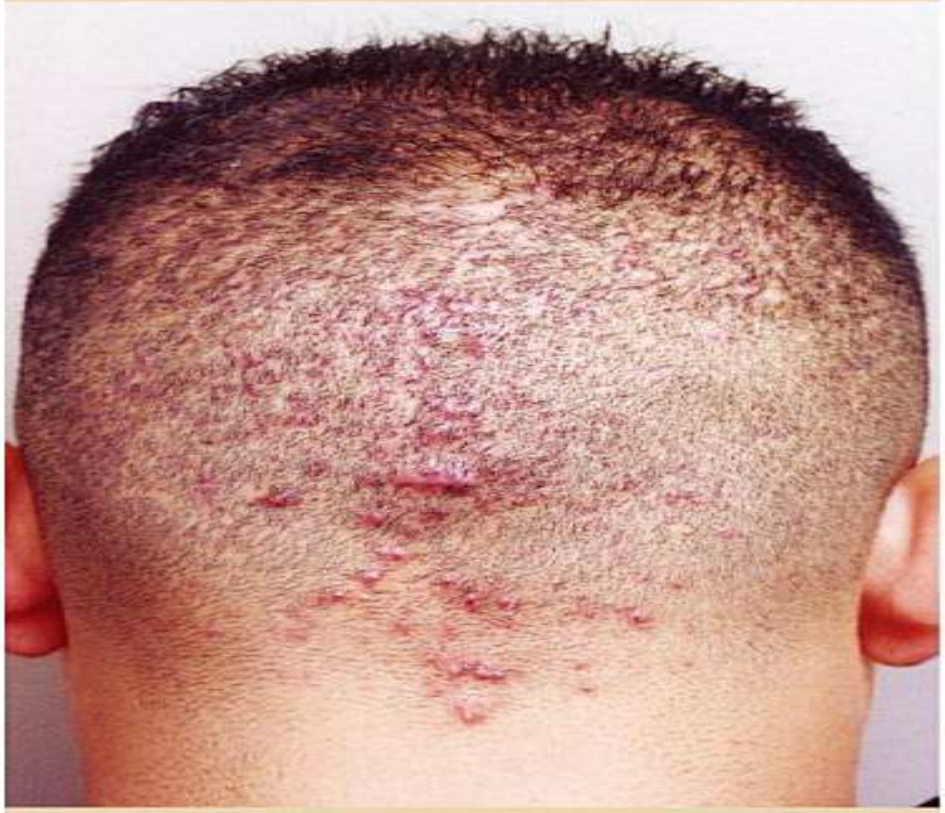


FIGURE 1-19 Acne keloidalis. (*Used with permission from Dr. Adelaide Hebert.*)

- **Histology:** follicular dilatation and mixed periinfundibular infiltrate with follicular rupture and foreign-body granulomas
- **Treatment:** systemic antibiotics, topical and/or intralesional steroids

Hypertrichosis and Hirsutism

- **Hypertrichosis** is defined as excessive hair growth anywhere on the body in either males or females. It is important to distinguish **hypertrichosis** from **hirsutism**, which is a term reserved for females who grow an excessive amount of terminal hairs in androgen-dependent sites.

Hypertrichosis

- There are several ways of classifying hypertrichosis. These are based on the distribution (**generalized vs. localized**), the age of onset (**congenital versus acquired**), and the type of hair (**vellus versus terminal**).

Treatment and prognosis

- **Treatment**

Laser hair removal, depilatory creams

- **Prognosis** : depending on the type of hypertrichosis. Hypertrichosis associated with genetic syndromes is generally lifelong.

drug-induced hypertrichosis, it is usually reversible with discontinuation of the medication

Hirsutism

Hirsutism is the growth of excessive male-pattern hair in women after puberty.

. It affects facial and body areas dependent on androgens, namely mustache and beard, pubic hair, buttocks, and thighs.

Treatment / Management

1 - Pharmacologic Treatment

Treatment of non-tumoral hirsutism still includes:

- oral contraception
- Spironolactone
- Finasteride

Treatment / Management

2- **Non-Pharmacologic Treatment**

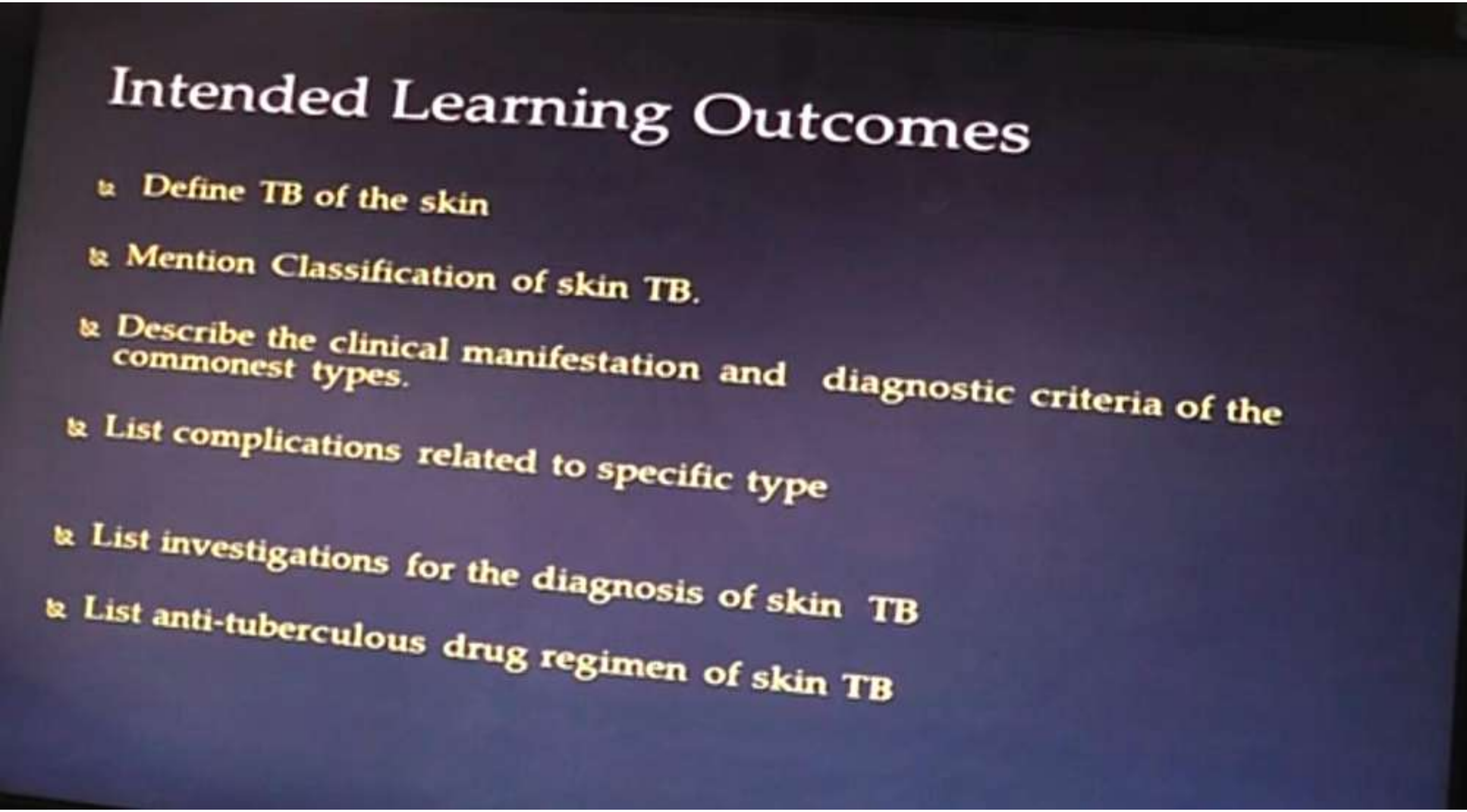
- the cosmetic approaches that are also appropriate for hypertrichoses: shaving, hair removal lasers and various intense pulsed light devices.

THANKS!

For more Question feel free to contact me
mobile: +967778444430
Email : almurieshmaher@gmail.com

SKIN TUBERCLEOSIS

Dr.belquis al-shaweri



Intended Learning Outcomes

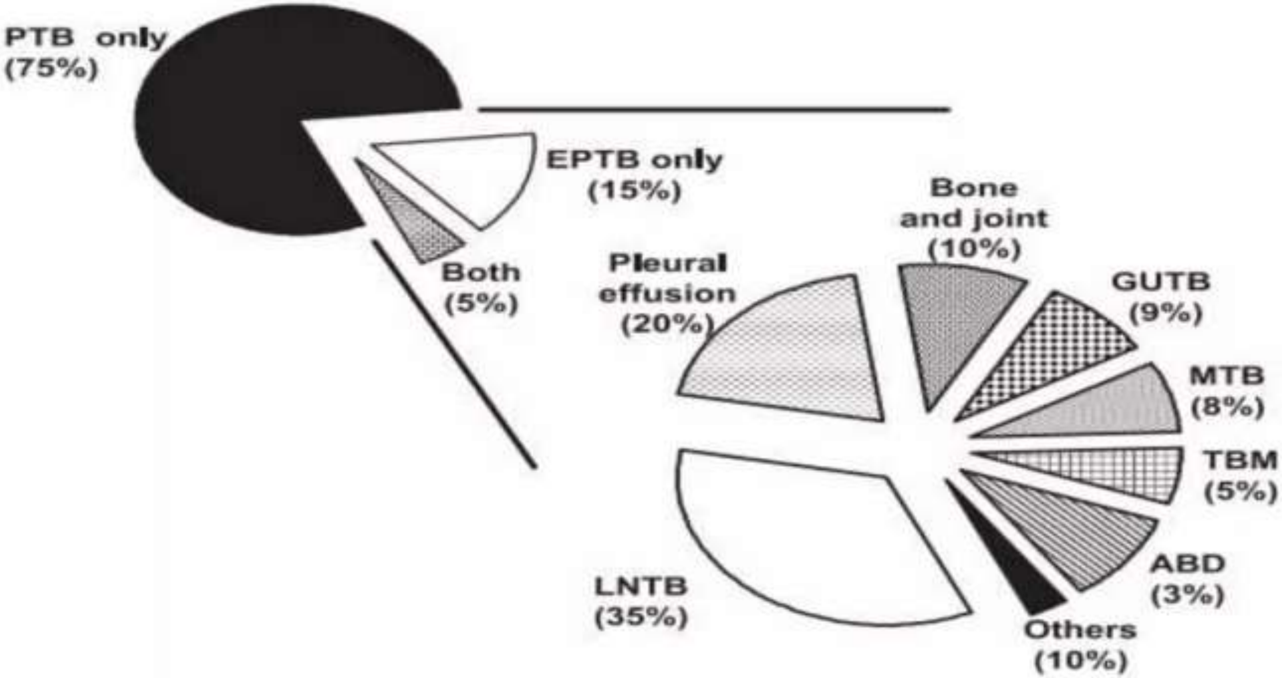
- Define TB of the skin
- Mention Classification of skin TB.
- Describe the clinical manifestation and diagnostic criteria of the commonest types.
- List complications related to specific type
- List investigations for the diagnosis of skin TB
- List anti-tuberculous drug regimen of skin TB

CAUSATIVE ORGANISM

- Mycobacterium tuberculosis
- Mycobacterium bovis
- BCG (Bacille calmette guerin)

Epidemiology

- Childhood TB
  - 5-15% of all cases of TB
  - most commonly in 10-14 years of age
- Commonest form
  - in adults: Lupus Vulgaris
  - in childhood: Scrofuloderma and Lichen scrofulosorum



CLASSIFICATION

Route of infection	Clinical type	histology	course
Inoculation tuberculosis (exogenous source)	Tuberculosis chancre	Non specific	Localized
	Tuberculosis verrucoasa cutis	TB specific	Localized
	Lupus vulgaris (occasionally)	TB specific	Localized
Secondary tuberculosis (endogenous source)			
Contiguous spread	Scrofuloderma	TB specific	Localized
Auto-inoculation	Orificial tuberculosis	TB specific	Progressive
Haematogenous tuberculosis	Acute military tuberculosis	TB specific	Generalized
	Lupus vulgaris	TB specific	Localized
	Tuberculous gumma	TB specific	Localized
Eruptive tuberculosis (Tuberculids)			
Micropapular	Lichen scrofulosorum	Variable	Localized
Papular	Papular or papulonecrotic tuberculid	variable	Scattered Crops
Nodular	Erythma induratum of Bazin	variable	Generalized

CLASSIFICATION

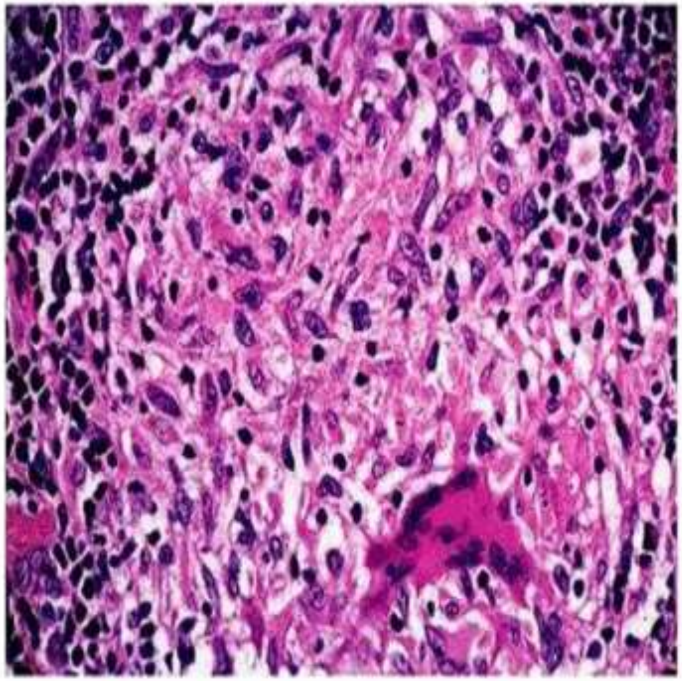
- Not previously exposed
  - Tuberculosis chancre
  - Milliary tuberculosis of the skin
- Previously sensitized hosts
  - Lupus vulgaris
  - Scrofuloderma
  - Tuberculosis verrucosa cutis

- Multibacillary
  - ❖ Primary inoculation TB, scrofuloderma, tuberculosis periorificialis, acute military tuberculosis, gumma
- Paucibacillary
  - ❖ Tuberculosis verrucosa cutis, lupus vulgaris

- Most patients show clinical infection within first 3 decades of life.
- Female preponderance in scrofuloderma & lupus vulgaris.
- M. bovis found in 1–1.5%
- Childhood TB
  - 5-15% of all cases of TB
  - most commonly in 10-14 years of age
- Commonest form
  - In adults: Lupus Vulgaris
  - In childhood: Scrofuloderma and Lichen scrofulosorum

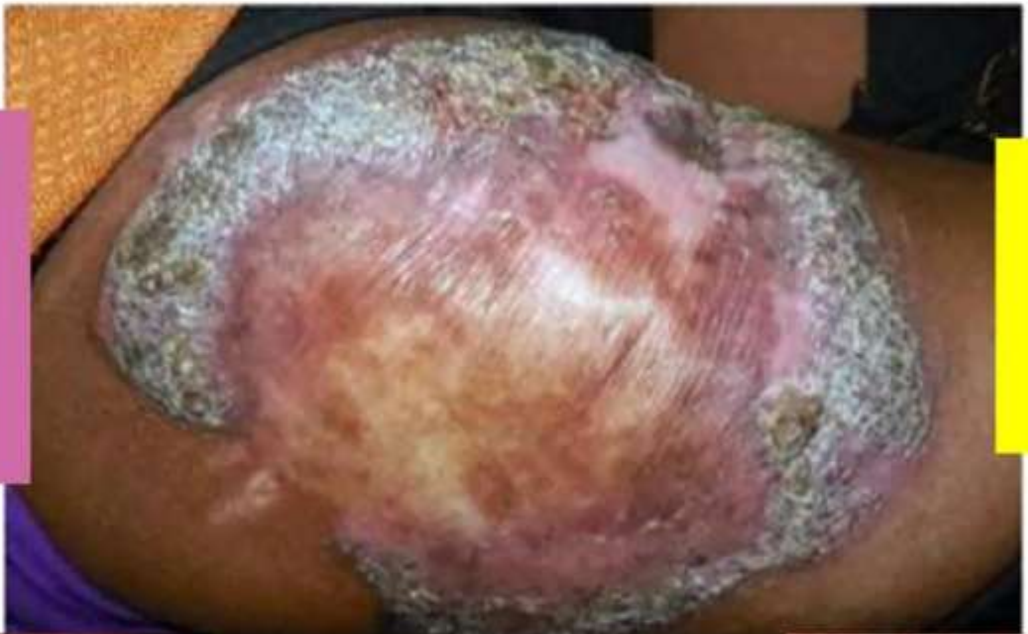
PATHOLOGY

- Tubercle→ an avascular granuloma composed of a central zone containing giant cells, with or without caseation, and a peripheral zone of lymphocyte and fibroblast

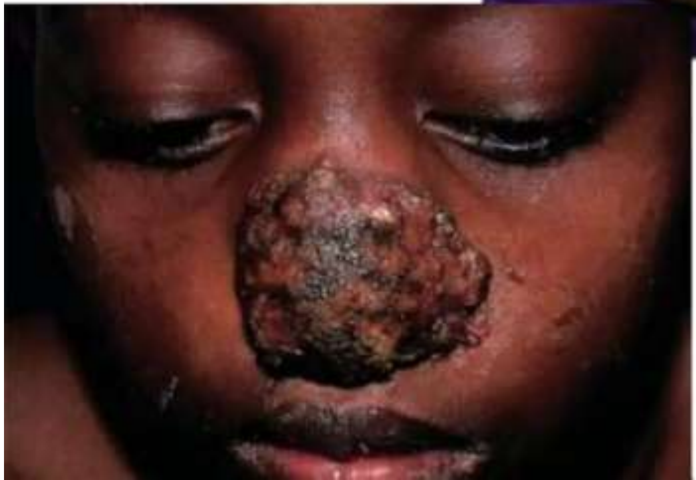


LUPUS VULGARIS

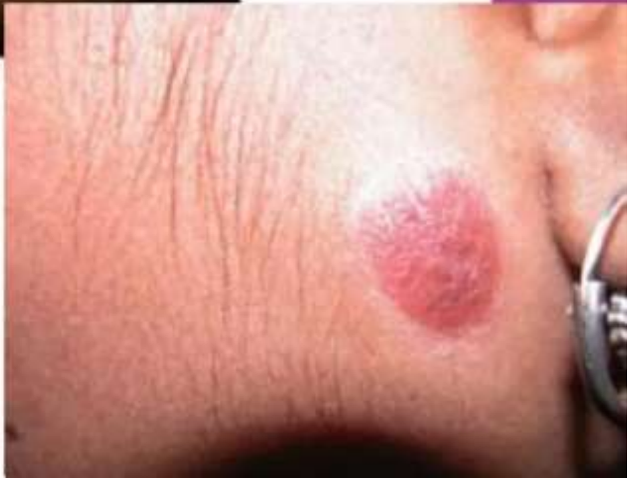
1808, **Robert willan**
Founder of british derma.
Gave term LUPUS
To nodular eruption on face

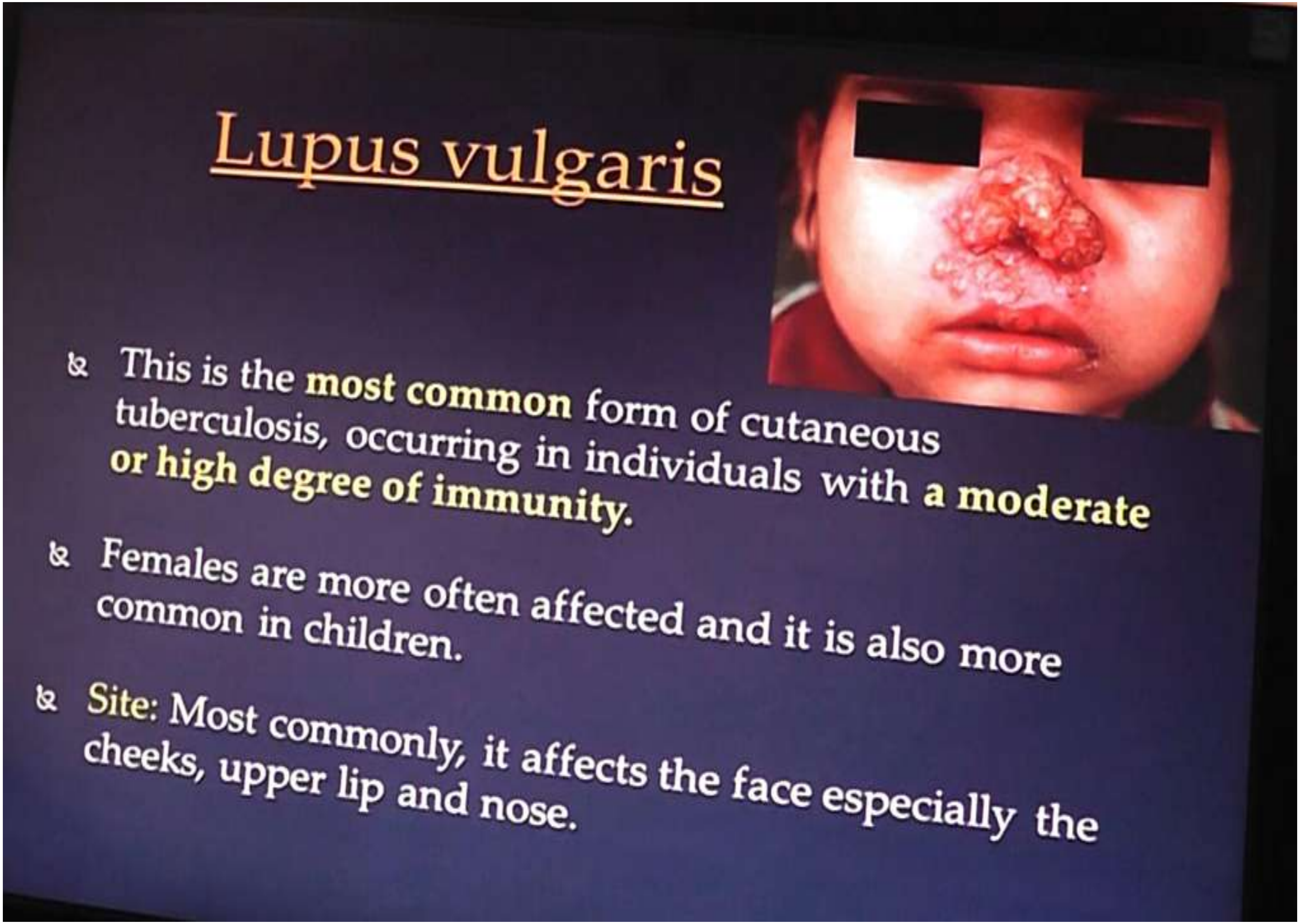


1887, **William Tilburg Fox**
used term LUPU
VLGARIS
for skin TB



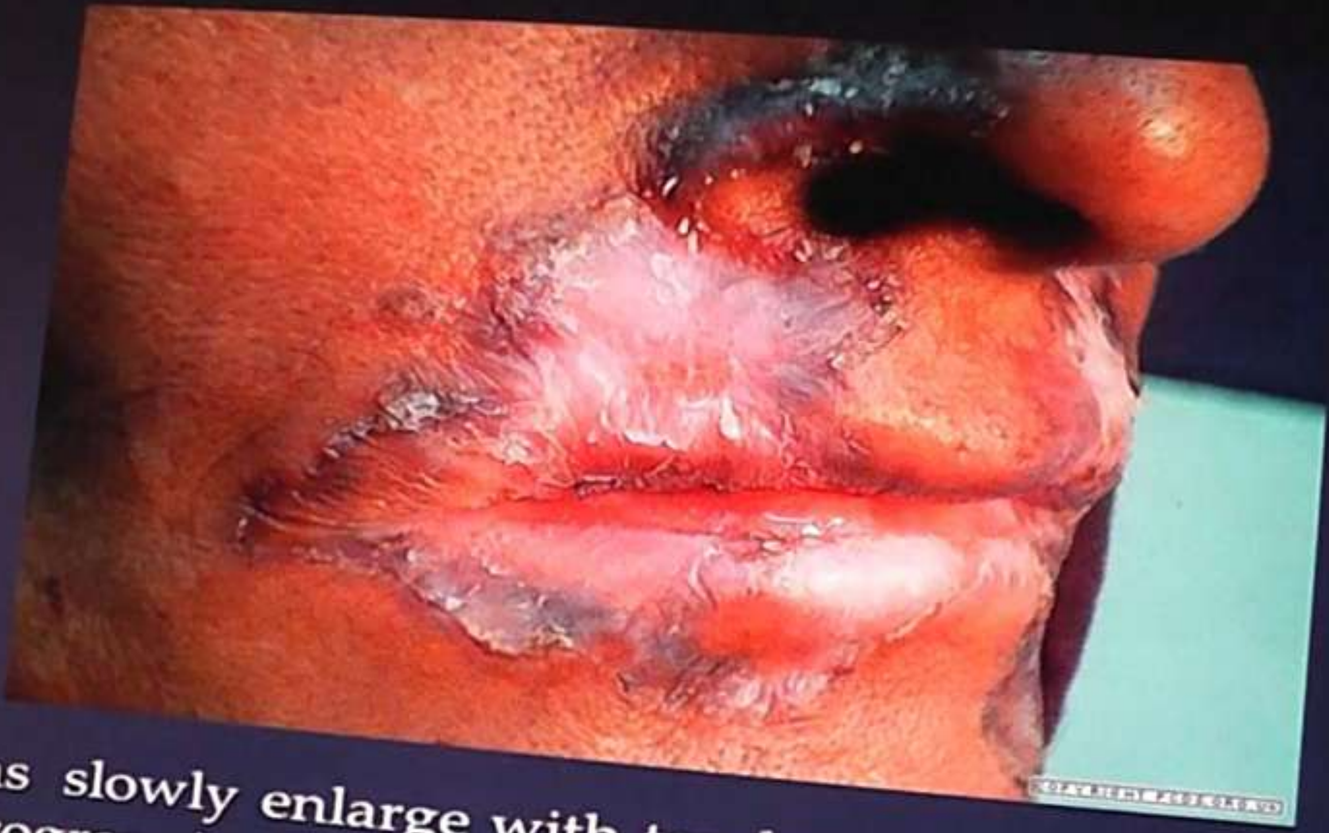
LUPUS word Origin
? Latin word of wolf
? Greek word lepros



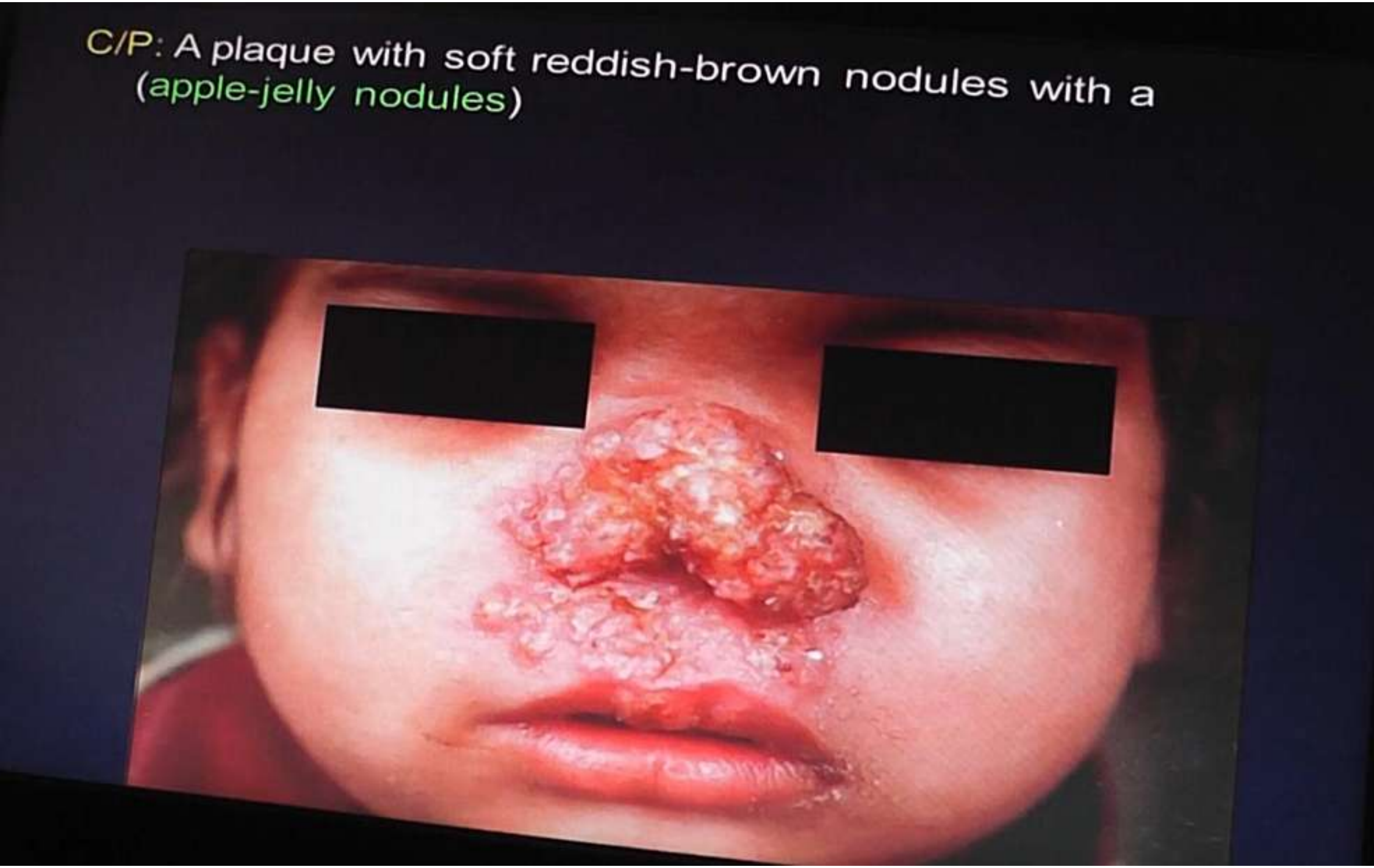


Lupus vulgaris

- This is the **most common** form of cutaneous tuberculosis, occurring in individuals with **a moderate or high degree of immunity.**
- Females are more often affected and it is also more common in children.
- **Site:** Most commonly, it affects the face especially the cheeks, upper lip and nose.

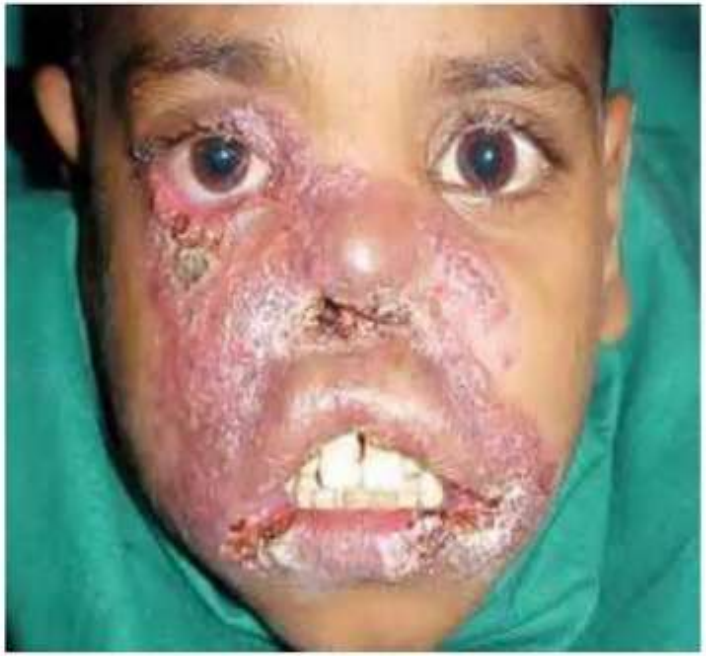


Lesions slowly enlarge with tendency to heal in one area and progress in another.



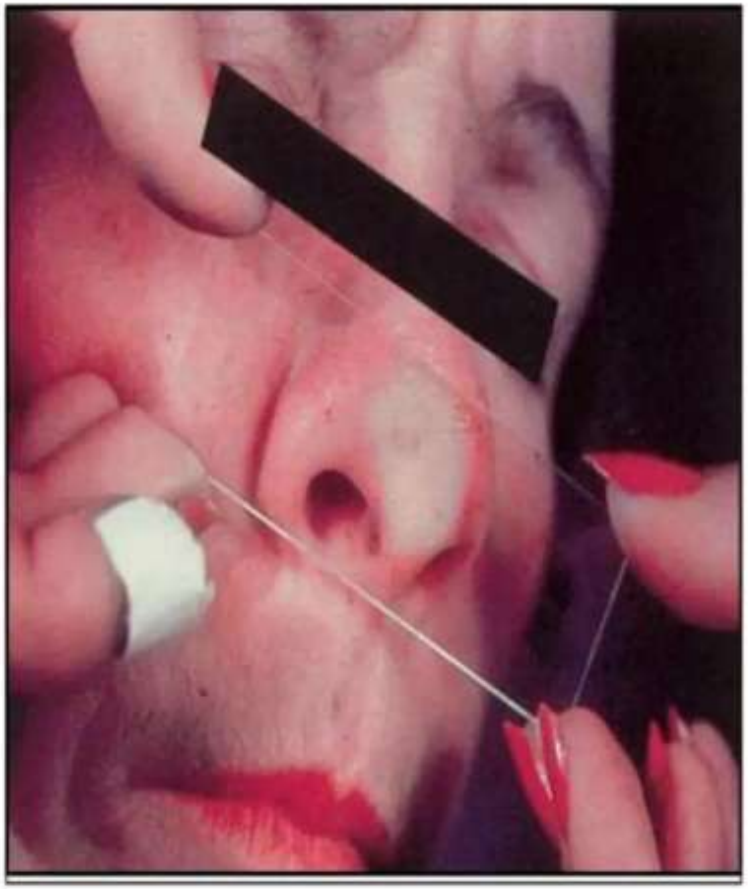
C/P: A plaque with soft reddish-brown nodules with a
(apple-jelly nodules)

- Mucosa- small, soft pink papule/ ulcer
- Nasal & auricular cartilage→ extensive destruction & disfigurement



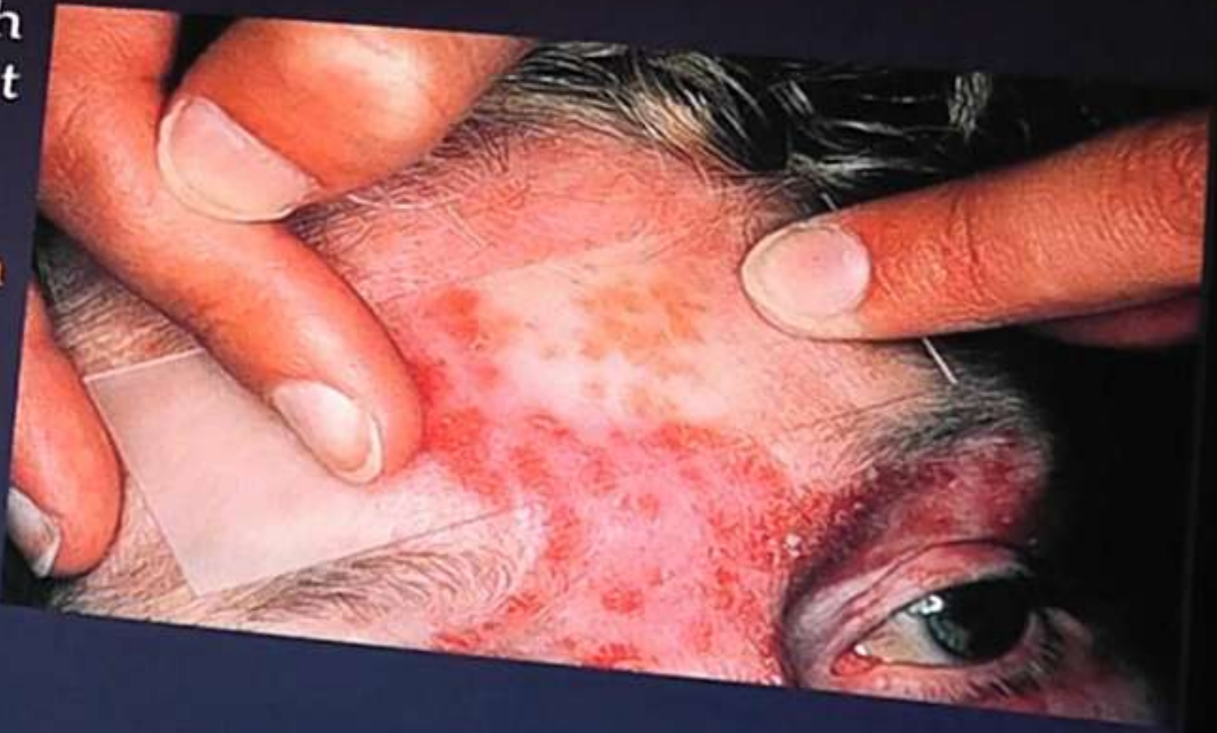
- **Diascopy test:**

- If lesion is pressed by a glass slide to diminish vascular component of inflammation, individual nodules appear as yellow brown spots (apple jelly color), so nodules are named “apple jelly nodules”.



Diascopy test

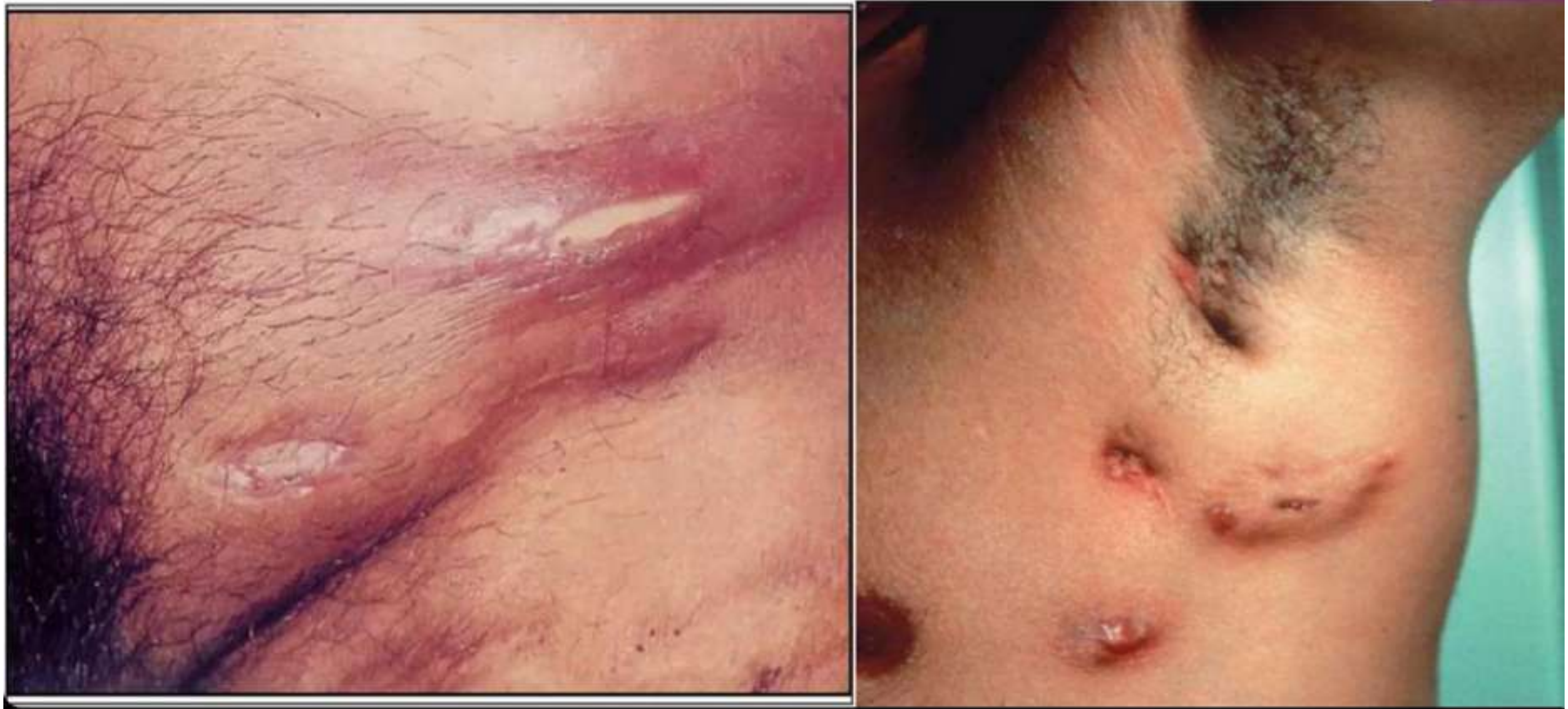
If the lesion is pressed by a glass slide to diminish the vascular component of inflammation, individual nodules appear as yellow brown spots (apple jelly color), so the nodules are named "apple jelly nodules".



SCROFULODERMA (TUBERCULOSIS COLLIQUATIVA CUTIS)

1883, **Ernest Besnier** wrote about scrofulous gumma

Latin word scrofa means a breeding sow, b/c swine were supposed to be subject to the complaint

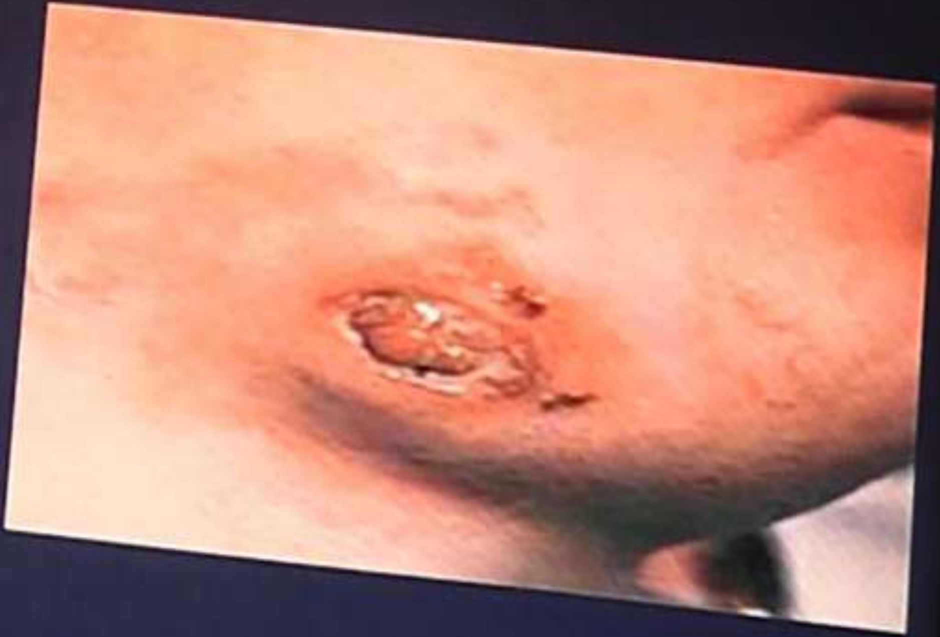


Scrofuloderma

It results as a direct extension from an underlying TB focus, such as

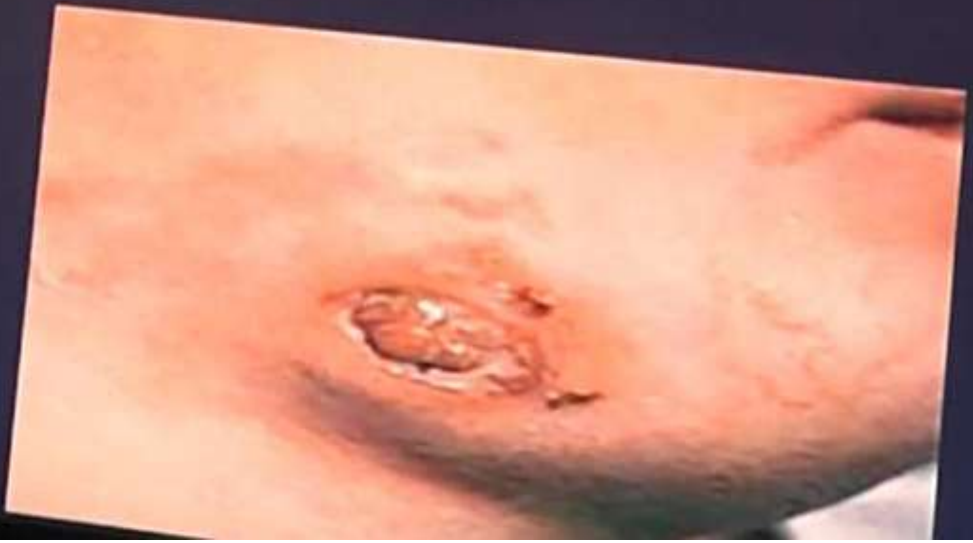
- ✓ a regional lymph node
- ✓ or infected bone
- ✓ or joint,

to the overlying skin.

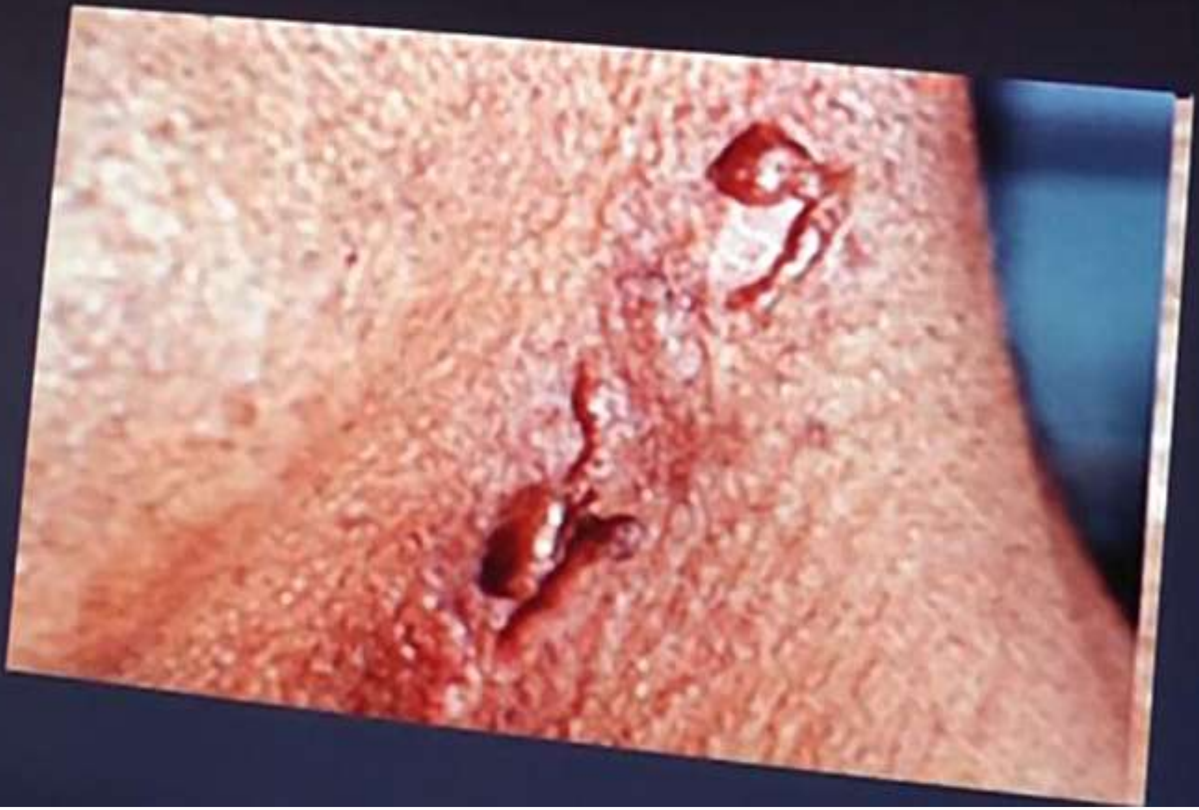


Scrofuloderma

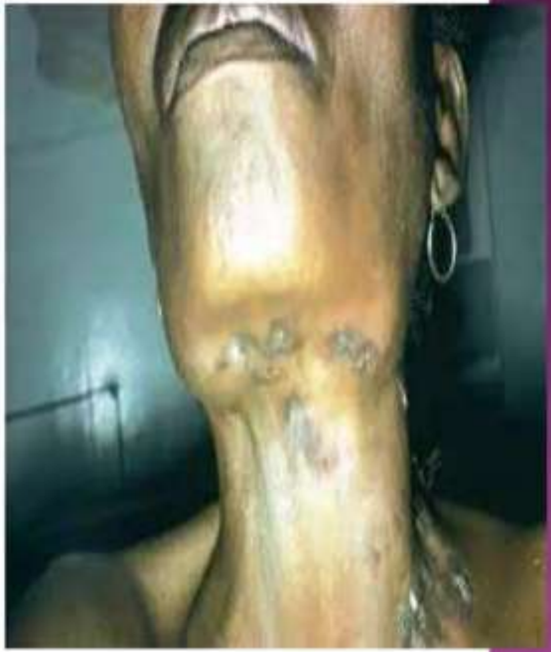
- C/P: presents with reddish blue induration of skin, overlying an infected focus, which suppurates and breaks down forming ulcers and sinus tracts that discharge purulent, or caseous material
- It causes fistulae and scarring



Combination of chronic discharging sinuses, hypertrophic scars, crusts, cicatritial bands is characteristic



- Scarring and fibrosis of lymph nodes may lead to lymphoedema and elephantiasis
- Numerous fistulae may intercommunicate beneath ridges of a bluish skin
- Skin of the lymphoedematous area may show lesions of cutaneous TB, usually lupus vulgaris
- Spontaneous healing with cribriform scarring possible



WARTY TUBERCULOSIS

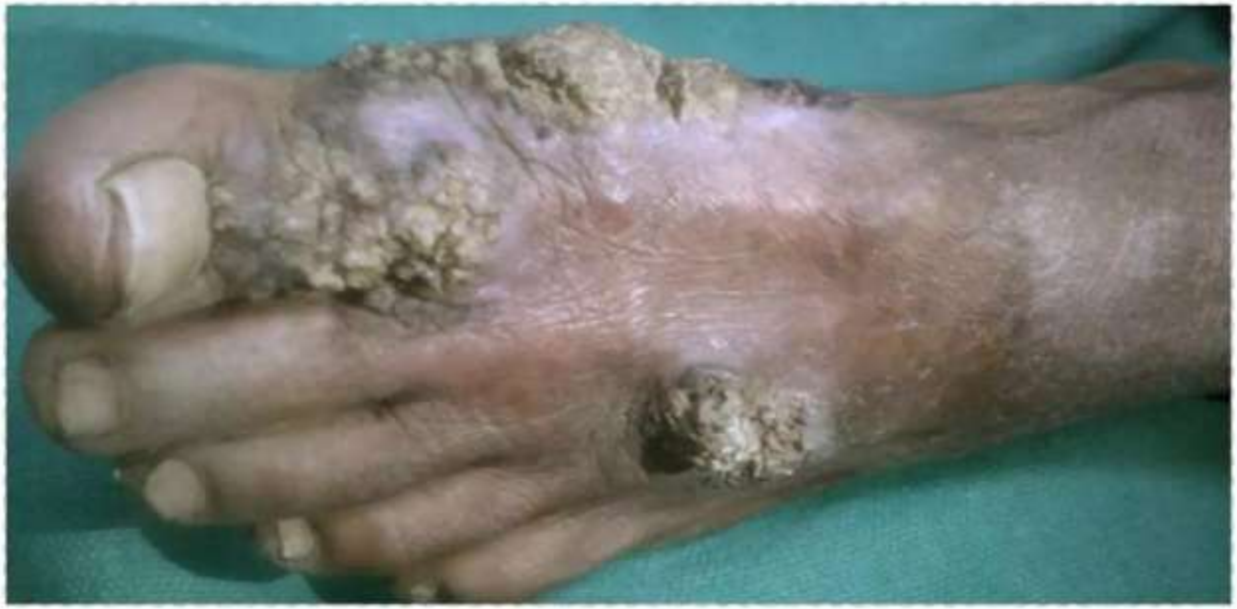
Synonyms:

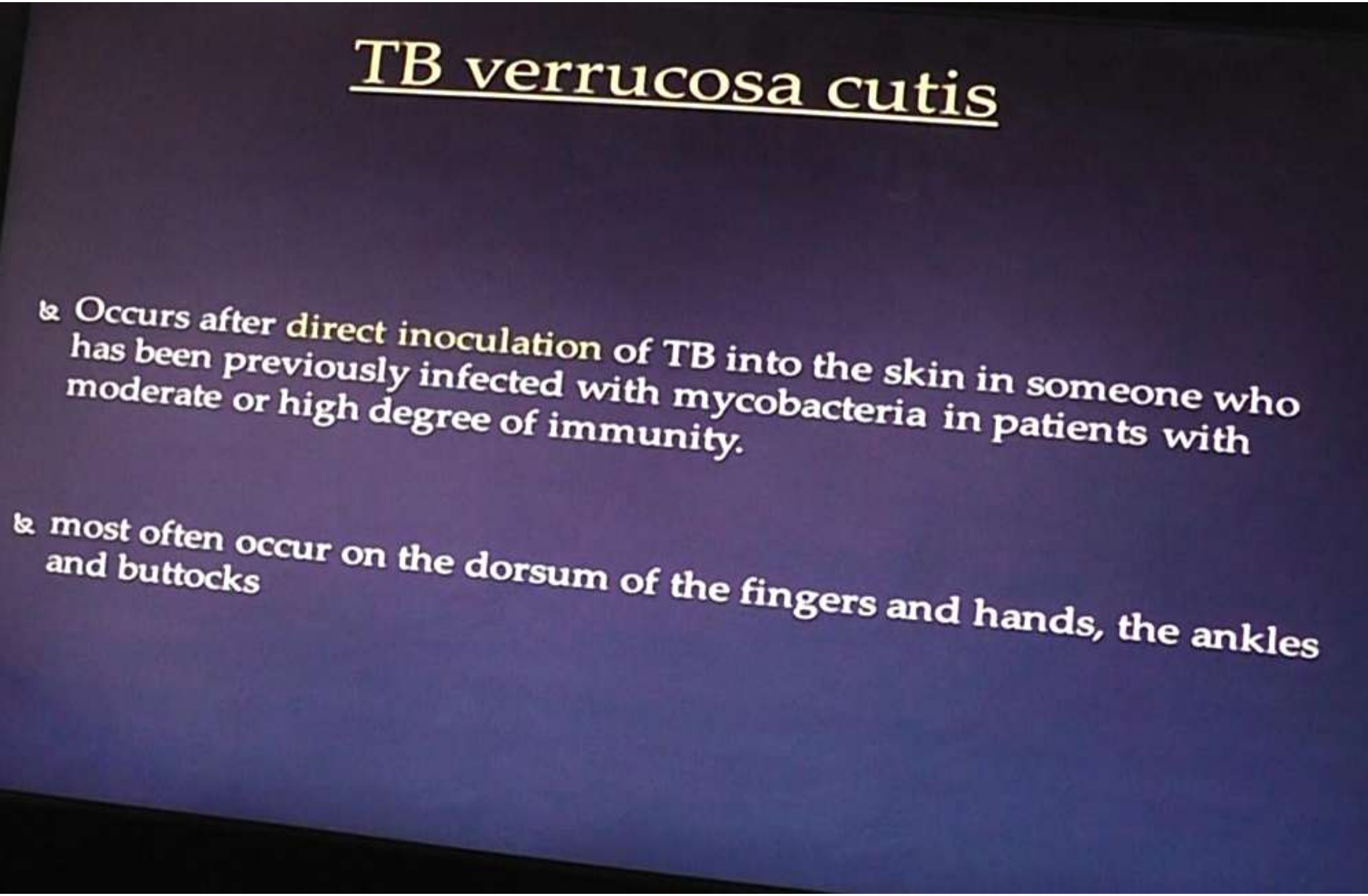
Tuberculosis verrucosa cutis

Anatomist’s warts

Prosector’s warts

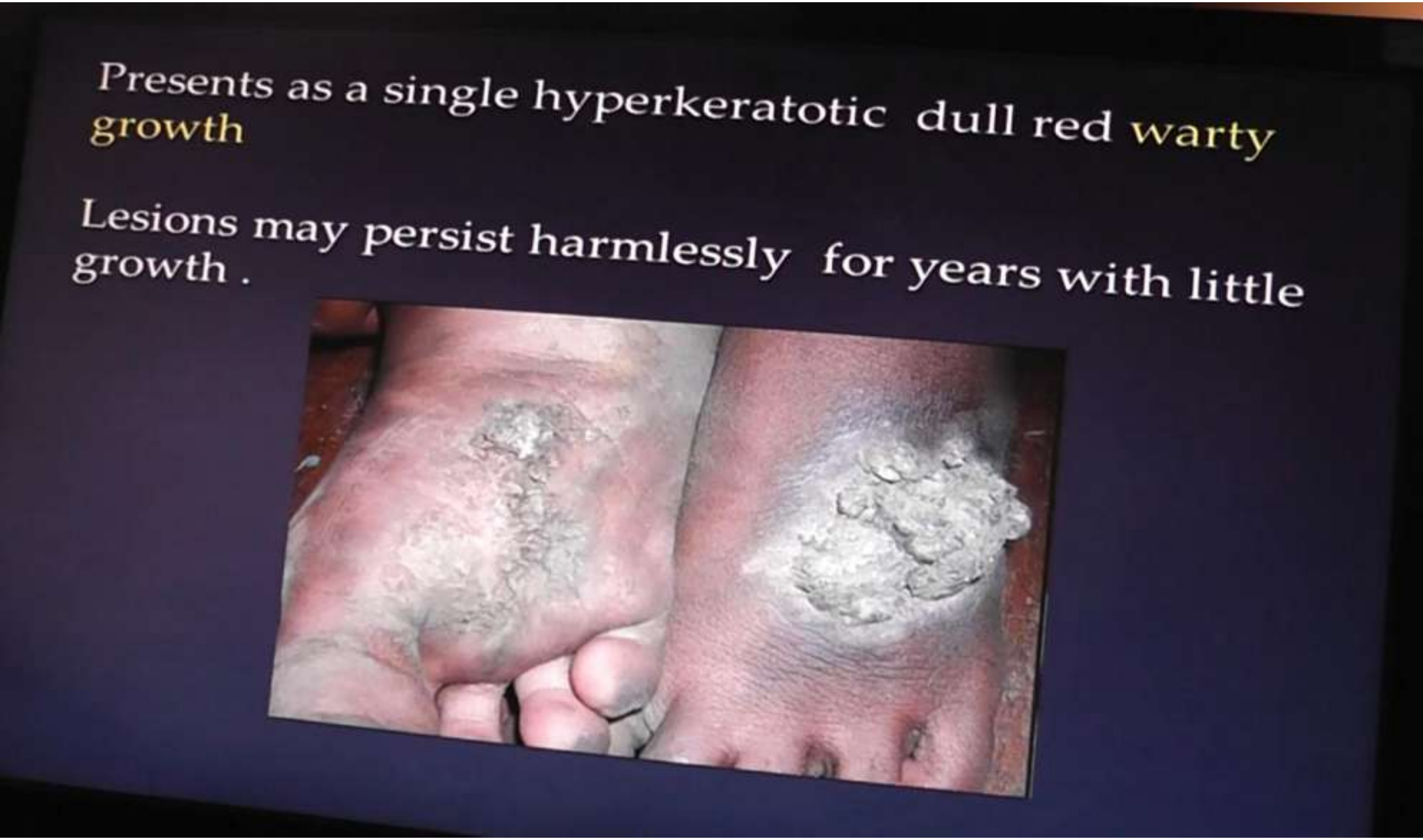
Verruca necrogenica





TB verrucosa cutis

- Occurs after direct inoculation of TB into the skin in someone who has been previously infected with mycobacteria in patients with moderate or high degree of immunity.
- most often occur on the dorsum of the fingers and hands, the ankles and buttocks



Presents as a single hyperkeratotic dull red warty growth

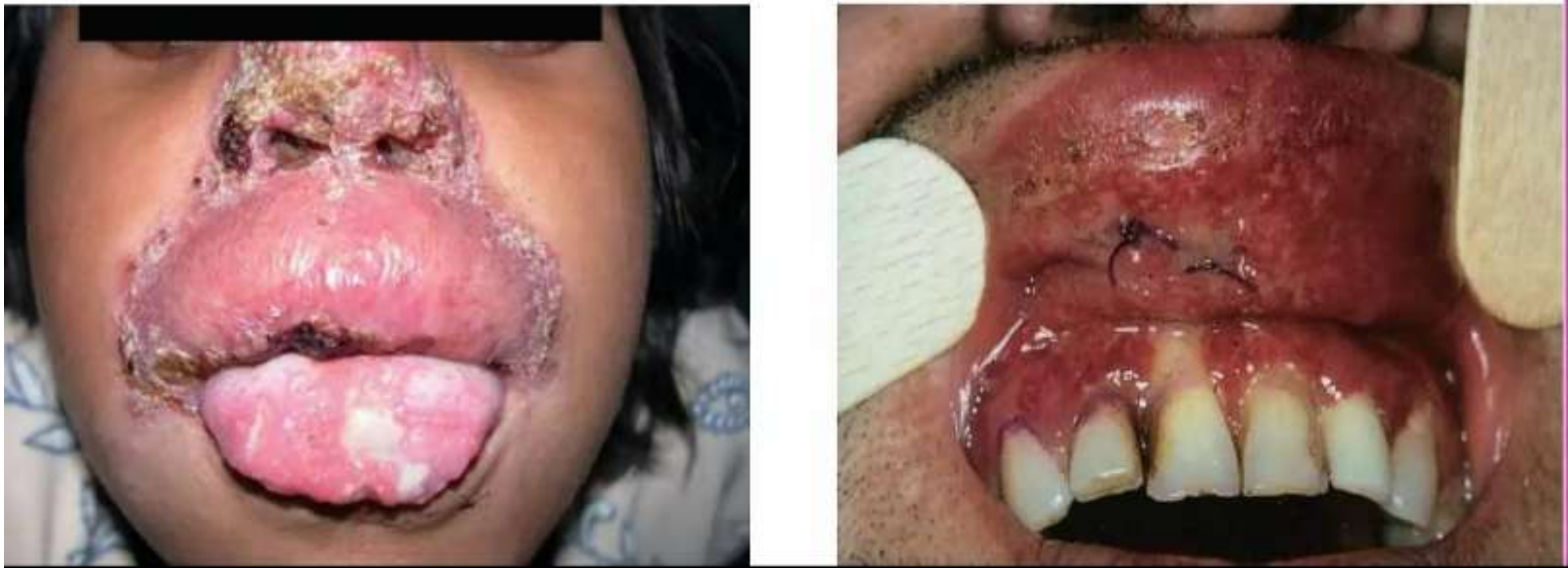
Lesions may persist harmlessly for years with little growth .

ACUTE MILIARY TUBERCULOSIS

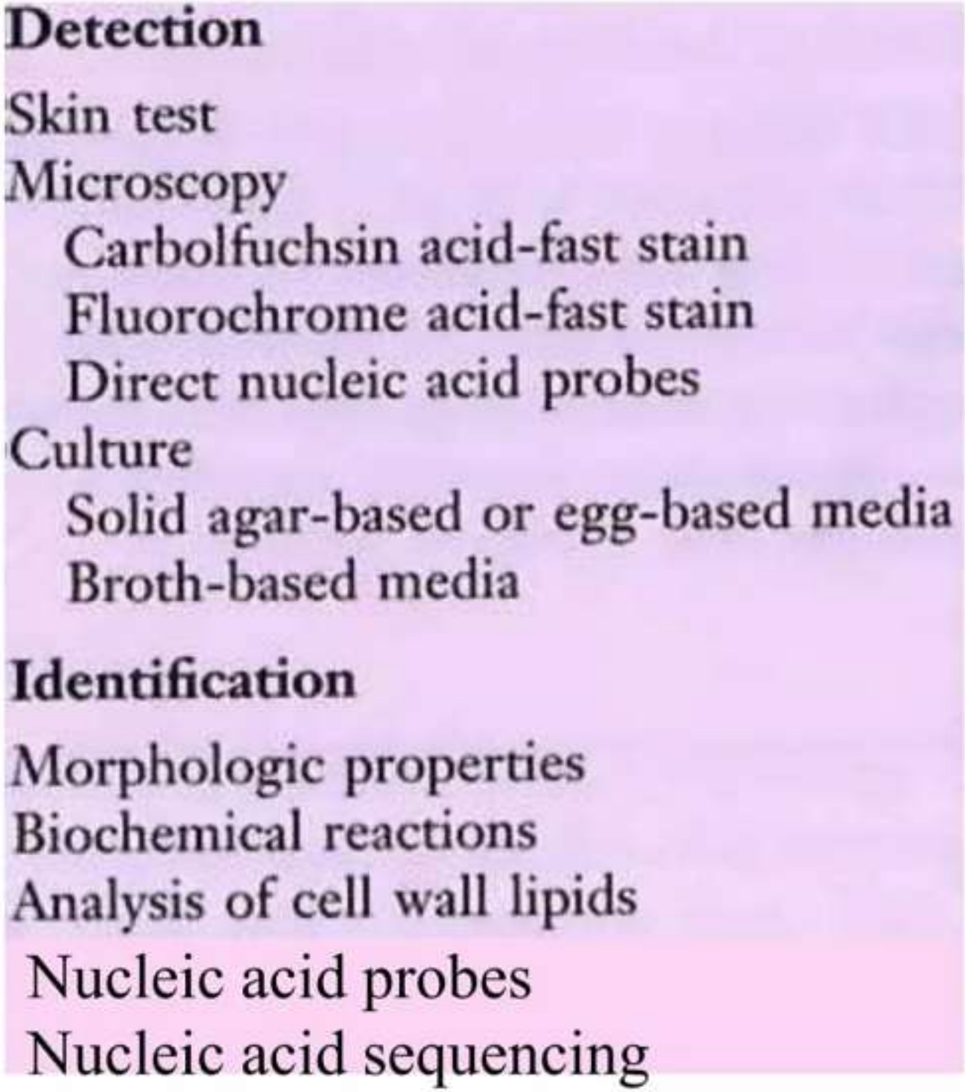
- Usually affects
  - Young children
  - Immunosuppressed patients
  - Concurrent HIV infection
- Crops of minute bluish papules, vesicles, pustules , nodules or haemorrhagic lesions
- Enlarged liver (40% of cases), enlarged spleen (15%), inflammation of the pancreas (<5%), and multiple organ dysfunction with adrenal insufficiency(adrenal glands do not produce enough steroid hormones to regulate organ function)

ORIFICIAL TUBERCULOSIS

- More common in males with impaired cell mediated immunity
- Site→ oral mucosa, anal mucosa & vulva
- Small oedematous red nodules rapidly break down to form painful, shallow ulcers with undermined bluish edges and hemorrhagic base.



LABORATORY DIAGNOSIS

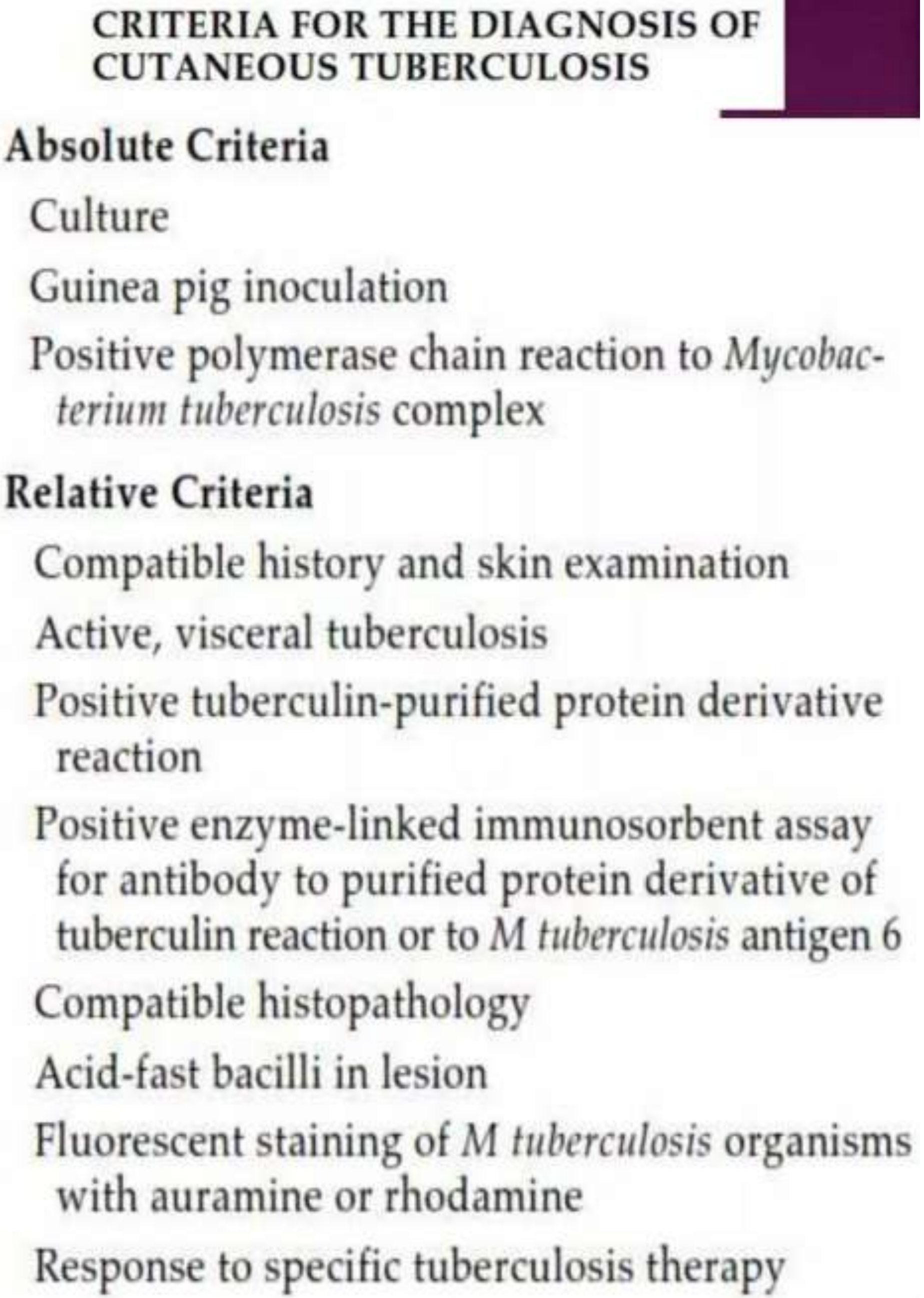


Detection

- Skin test
- Microscopy
  - Carbolfuchsin acid-fast stain
  - Fluorochrome acid-fast stain
  - Direct nucleic acid probes
- Culture
  - Solid agar-based or egg-based media
  - Broth-based media

Identification

- Morphologic properties
- Biochemical reactions
- Analysis of cell wall lipids
- Nucleic acid probes
- Nucleic acid sequencing



CRITERIA FOR THE DIAGNOSIS OF CUTANEOUS TUBERCULOSIS

Absolute Criteria

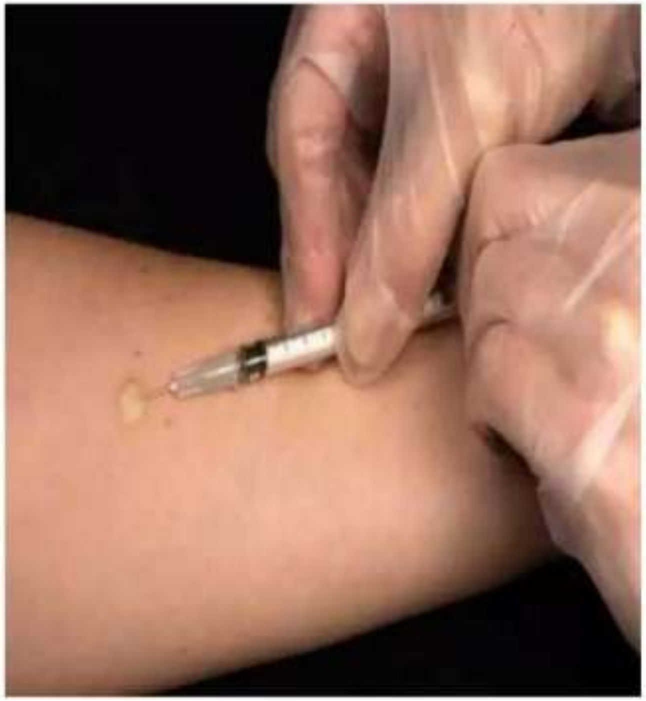
- Culture
- Guinea pig inoculation
- Positive polymerase chain reaction to *Mycobacterium tuberculosis* complex

Relative Criteria

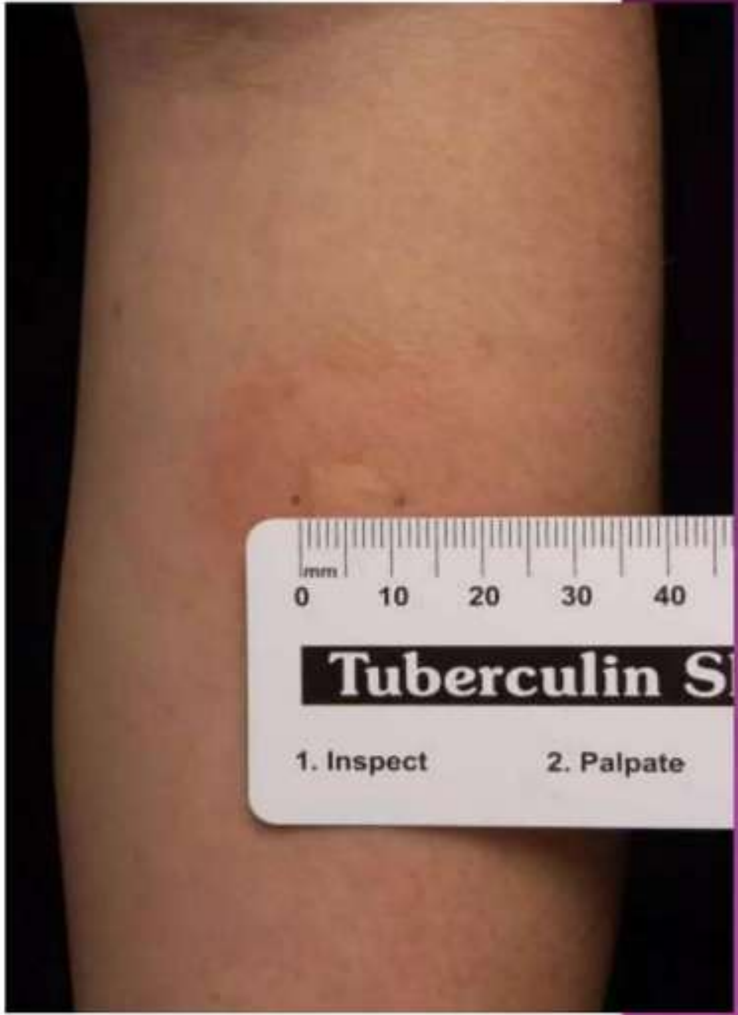
- Compatible history and skin examination
- Active, visceral tuberculosis
- Positive tuberculin-purified protein derivative reaction
- Positive enzyme-linked immunosorbent assay for antibody to purified protein derivative of tuberculin reaction or to *M tuberculosis* antigen 6
- Compatible histopathology
- Acid-fast bacilli in lesion
- Fluorescent staining of *M tuberculosis* organisms with auramine or rhodamine
- Response to specific tuberculosis therapy

MANTOUX TUBERCULIN SKIN TEST

- Tuberculin is made from proteins derived from inactive tubercle bacilli
- Most people who have TB infection will have a reaction at injection site
- 0.1 ml of 5 tuberculin units of liquid tuberculin are injected between the layers of skin on forearm



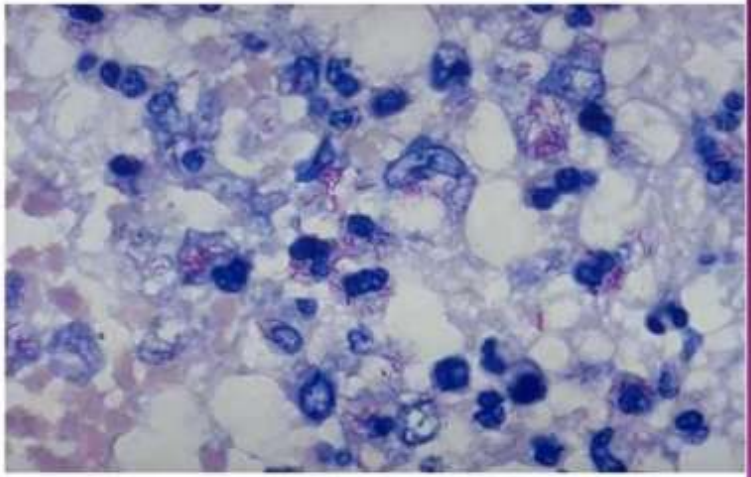
- Forearm should be examined within 48 - 72 hours
- Reaction is an area of induration (swelling) around injection site
  - Induration is measured in millimeters
  - Erythema (redness) is not measured

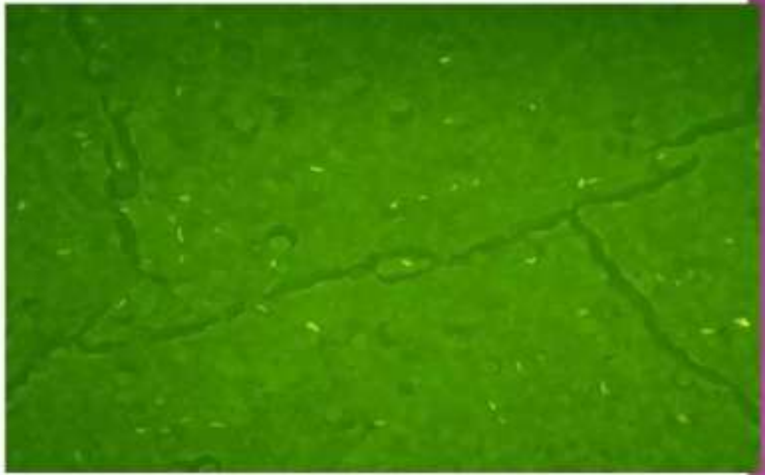

mm
0
10
20
30
40
Tuberculin S
1. Inspect
2. Palpate

Only the induration is measured

DIRECT MICROSCOPY IDENTIFICATION

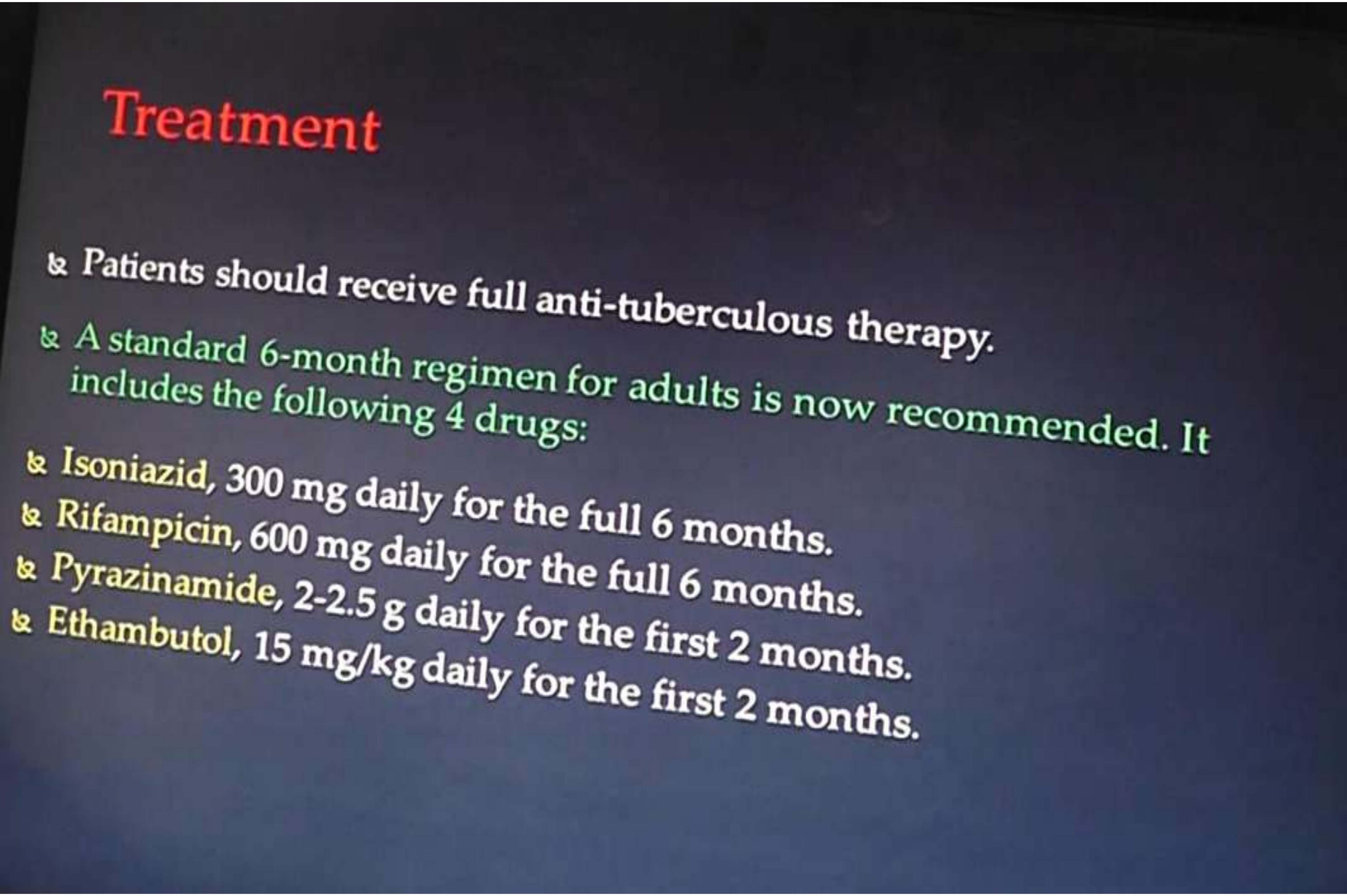
- Ziehl-Neelsen stain is used most commonly to demonstrate the presence of the bacilli in a smear. The technique is simple and inexpensive
- Fluorescent dye (Auramine O and Rhodamine B)





FIRST LINE DRUGS

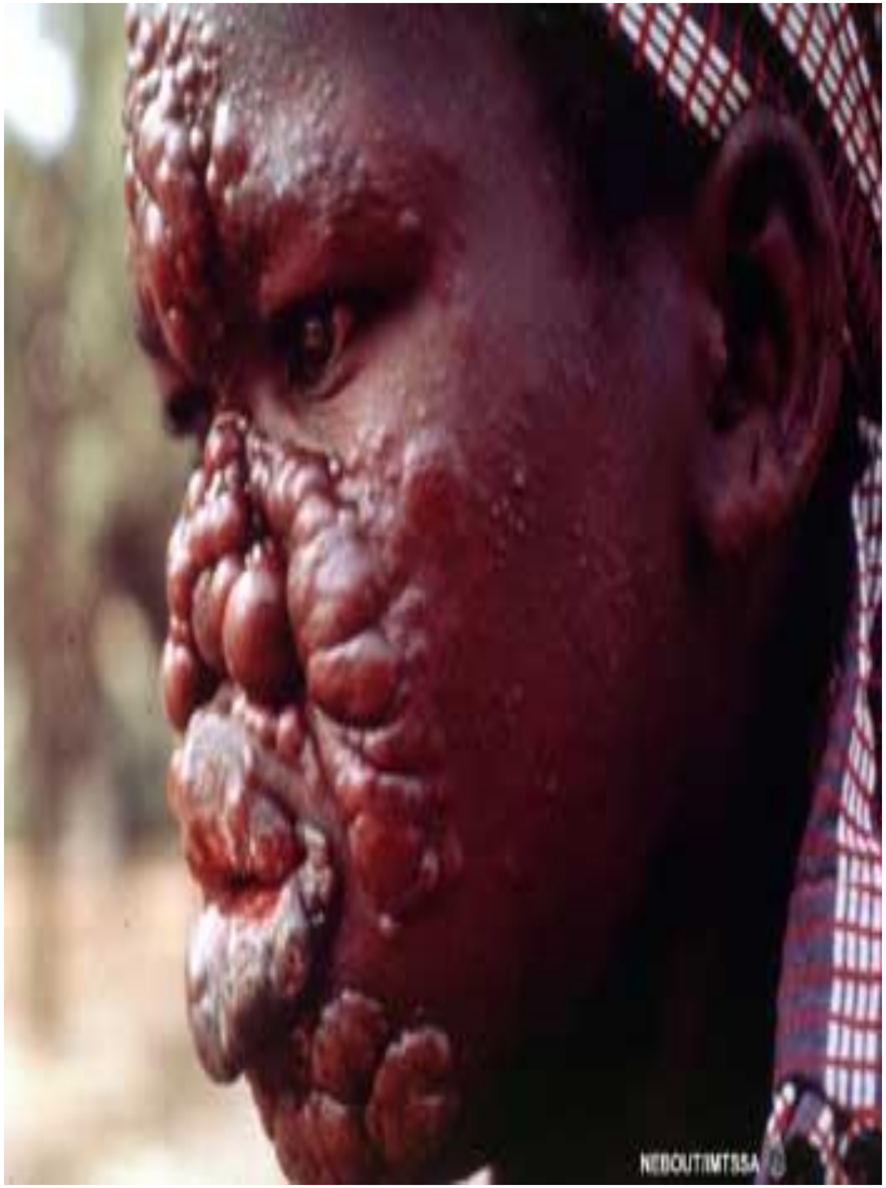
- Rifampicin(R) – Bactericidal, all bacilli
- Pyrazinamide(Z) – Bactericidal, all bacilli
- Isoniazid(H) – Bactericidal, replicating bacilli
- Streptomycin(S) – Bactericidal, Extracellular bacilli
- Ethambutol(E) - Bacteristatic



Treatment

- Patients should receive full anti-tuberculous therapy.
- A standard 6-month regimen for adults is now recommended. It includes the following 4 drugs:
- Isoniazid, 300 mg daily for the full 6 months.
- Rifampicin, 600 mg daily for the full 6 months.
- Pyrazinamide, 2-2.5 g daily for the first 2 months.
- Ethambutol, 15 mg/kg daily for the first 2 months.

LEPROSY



NEBOUTIMTSSA

LEPROSY

(HANSEN'S DISEASE)

1873

BACTERIA

MYCOBACTERIUM LEPRAE

GRAM POSITIVE OBLIGATE INTRACELLULAR

BACILLI

AEROBIC

NON MOTILE

NEVER GROW IN

VITRO

Leprosy (Hansen's Disease)

- Infectious disease caused by **obligate intracellular bacteria** *Mycobacterium leprae* and *Mycobacterium lepromatosis*
  - *Acid-fast bacilli*

Transmission

- 1) From untreated & infected patient's *nasal secretions* and *skin lesions*
- 2) Contact with *Armadillos* of the species *Dasypus novemcinctus* (nine-banded armadillo)

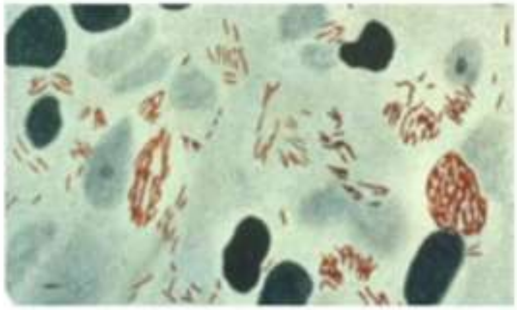
Risk Factors

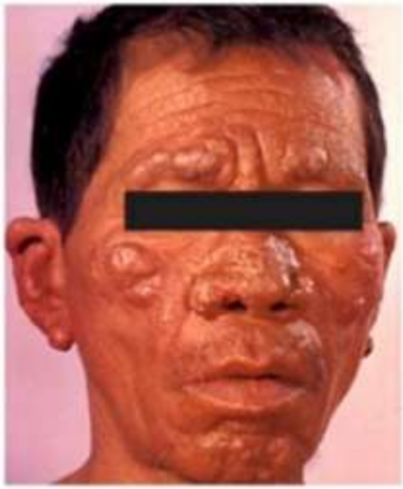
- 1) Close Contact
- 2) Age: Bimodal effect
  - Between ages 5-15 and older than 30 years old
- 3) Genetics
  - *NOD2 variants*
- 4) Immunosuppression
  - *Chemotherapy, HIV positive*

Leprosy: Pathogenesis

- Invades through epithelium and into peripheral nerves
- *Obligate intracellular parasite that lives within macrophages*
- *Slow growing organism*
    - *Multiplies in approximately 12.5 days*
- Growth most efficient and fastest at temperatures of 27-33 degrees celsius
    - ***Affects coolest parts of body the most*** (skin, eyes, nose, mucous membranes of respiratory tract)

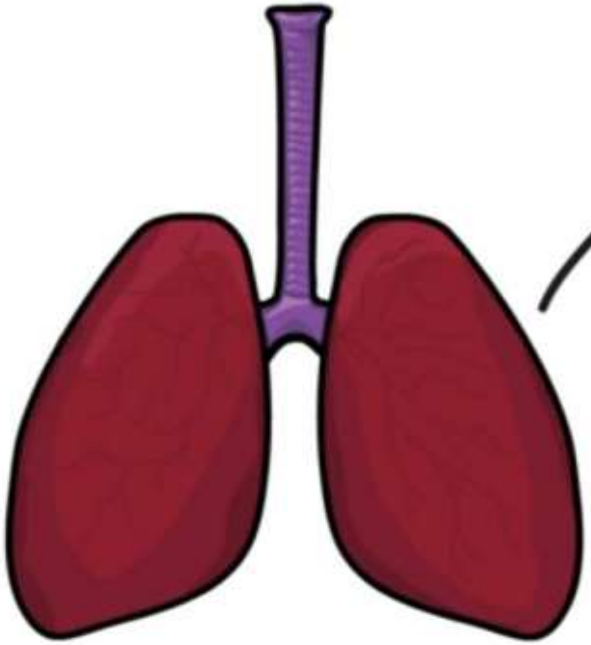
Most individuals do not become infected after exposure
When infected, causes chronic granulomatous disease

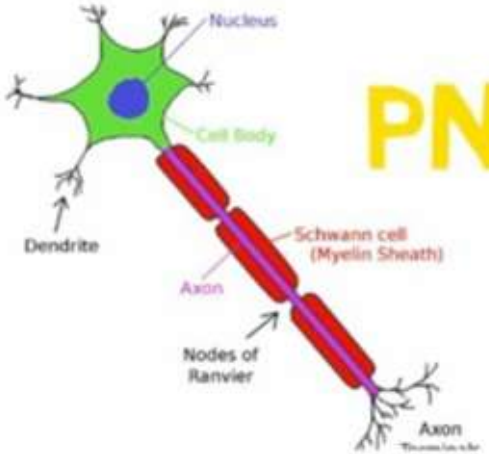




TRANSMISSION AND PATHOGENESIS

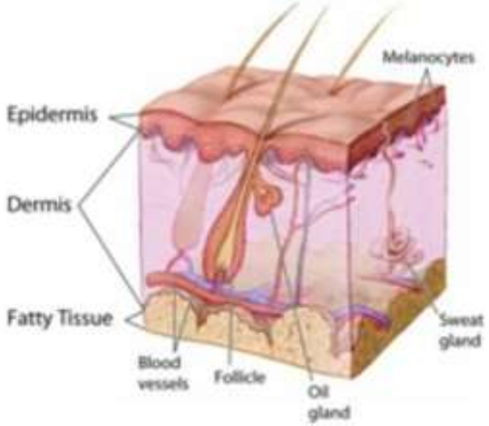
TRANSMISSION: INHALATION




Nucleus
Cell Body
Dendrite
Schwann cell
(Myelin Sheath)
Axon
Nodes of Ranvier
Axon

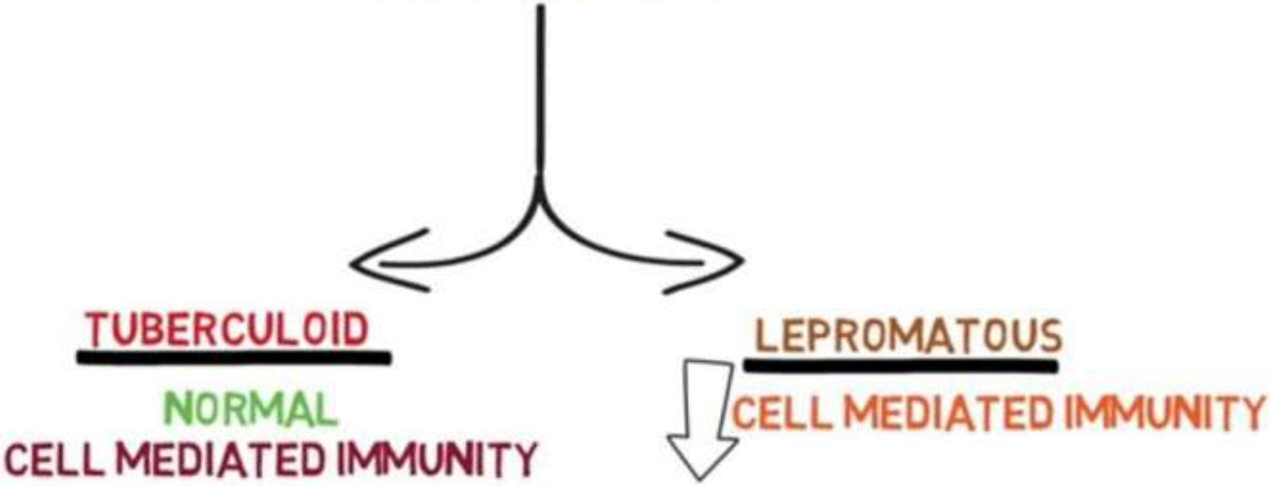
PNS

HEMATOGENESIS SPREAD


Melanocytes
Epidermis
Dermis
Fatty Tissue
Blood vessels
Follicle
Oil gland
Sweat gland

PATHOGENESIS

LEPROSY



TUBERCULOID

NORMAL
CELL MEDIATED IMMUNITY

LEPROMATOUS

↓ CELL MEDIATED IMMUNITY

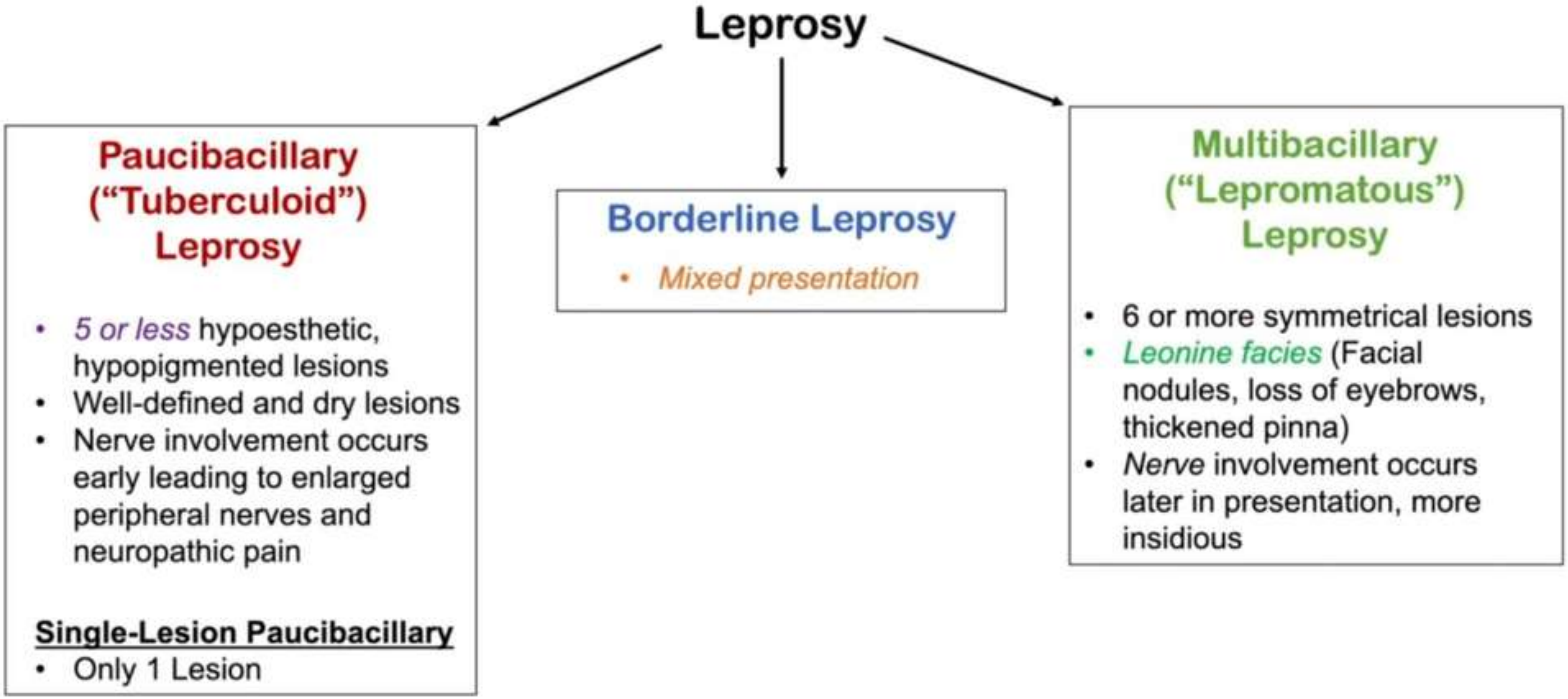
BORDERLINE LEPROSY

MODERATE
CELL MEDIATED IMMUNITY

VERY LOW
LEPROMATOUS

NORMAL
TUBERCU

Leprosy: Subtypes



Leprosy

Paucibacillary (“Tuberculoid”) Leprosy

- *5 or less* hypoesthetic, hypopigmented lesions
- Well-defined and dry lesions
- Nerve involvement occurs early leading to enlarged peripheral nerves and neuropathic pain

Single-Lesion Paucibacillary

- Only 1 Lesion

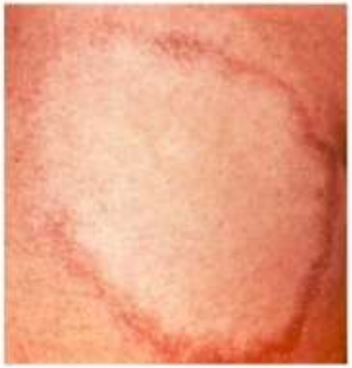
Borderline Leprosy

- *Mixed presentation*

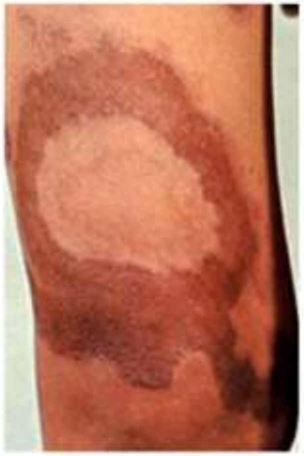
Multibacillary (“Lepromatous”) Leprosy

- 6 or more symmetrical lesions
- *Leonine facies* (Facial nodules, loss of eyebrows, thickened pinna)
- *Nerve* involvement occurs later in presentation, more insidious

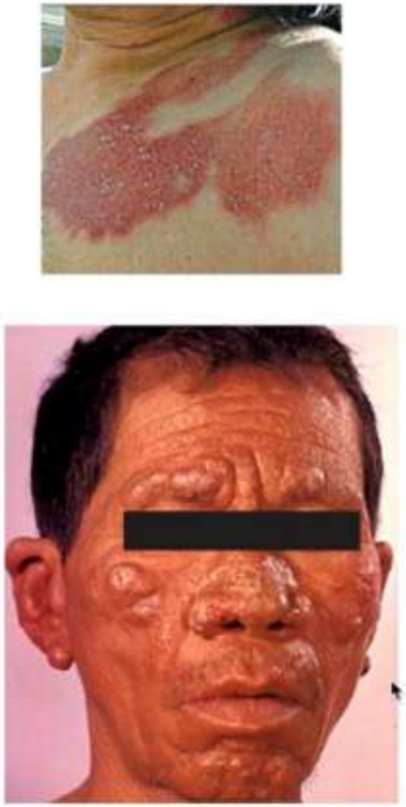
Paucibacillary Leprosy



Borderline Leprosy



Multibacillary Leprosy

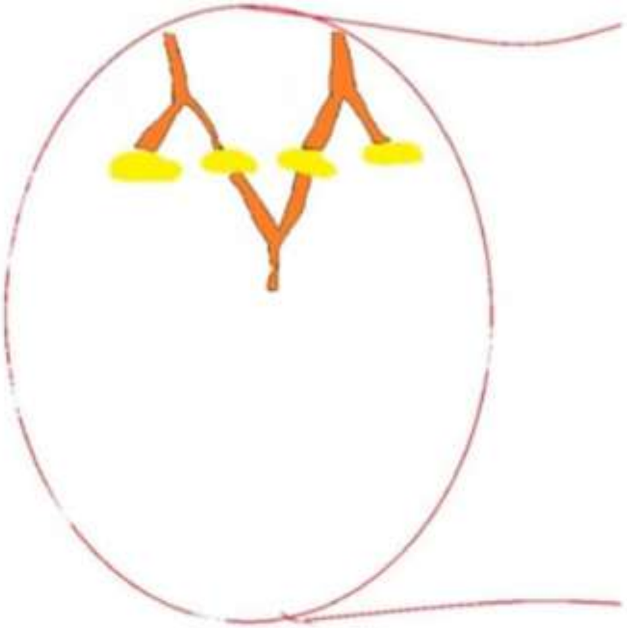


LEPROMATOUS LEPRASY

TYPE 3 HYPERSENTIVITY

NO CELL MEDIATED IMMUNITY

ANTIGEN ANTIBODY COMPLEX

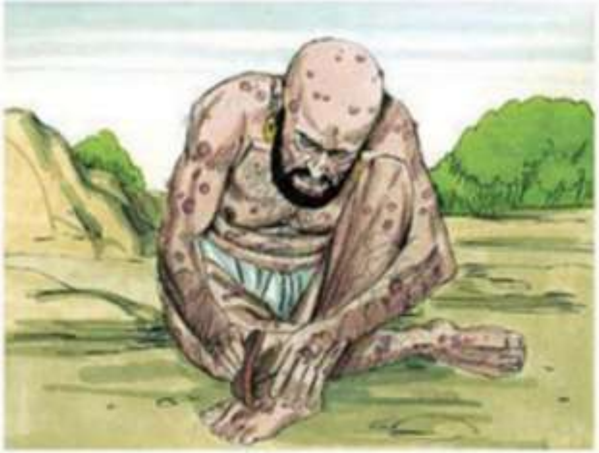


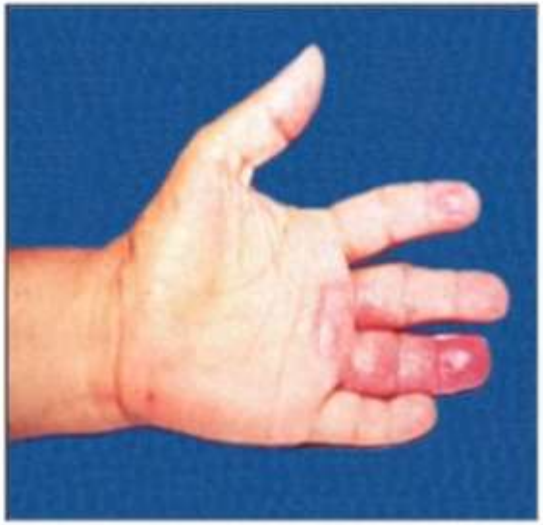
MULTI ORGAN INVOLVEMENT

	INCUBATION
TUBERCULOID LEPRASY	8-12 YEARS
LEPROMATOUS LEPRASY	2-5 YEARS

CLINICAL

	LEPROMATOUS	TUBERCULOID
SKIN LESION DISTRIBUTION	THROUGHOUT BODY	FEW SITES

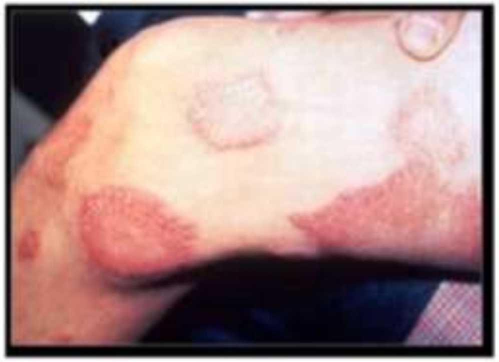




CLINICAL

	LEPROMATOUS	TUBERCULOID
SKIN LESION DISTRIBUTION	THROUGHOUT BODY	FEW SITES

SKIN LESION



- HYOPIGMENTED
- MACULES OR PLAQUES
- ANNULAR SHAPE

- NO SENSATION
- NO HAIR
- NO SWEATING

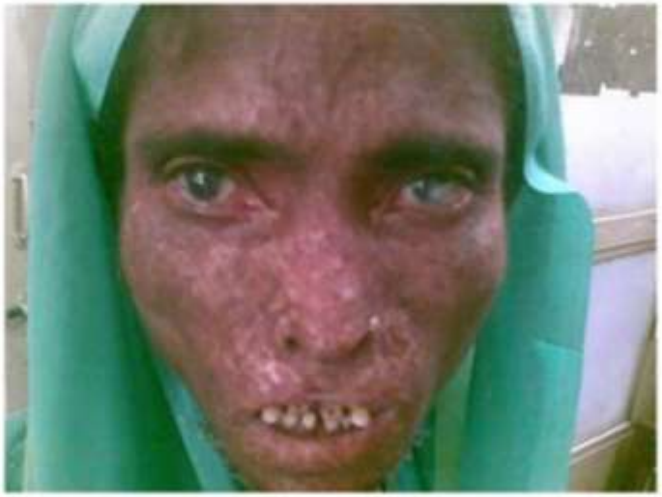
LEPROMATOUS

LION FACE

FACIAL SKIN THICKING

LEONINE FACIES

LOSS OF NASAL SEPTUM

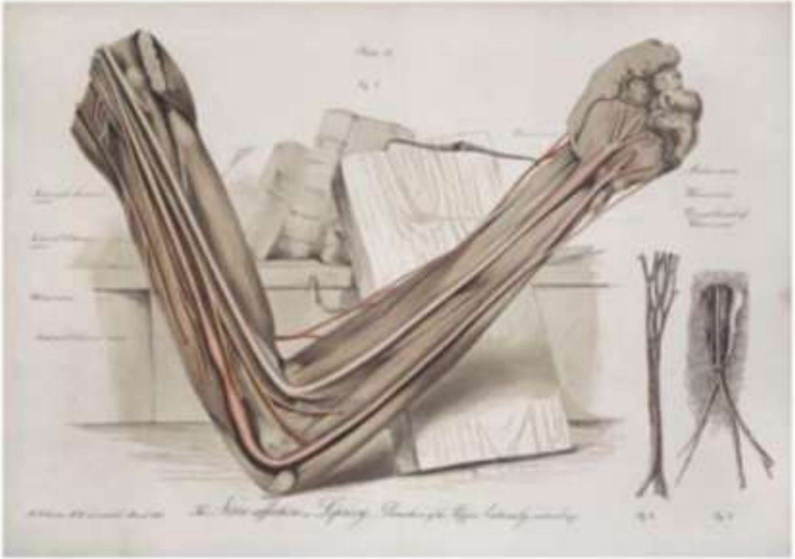


NERVE

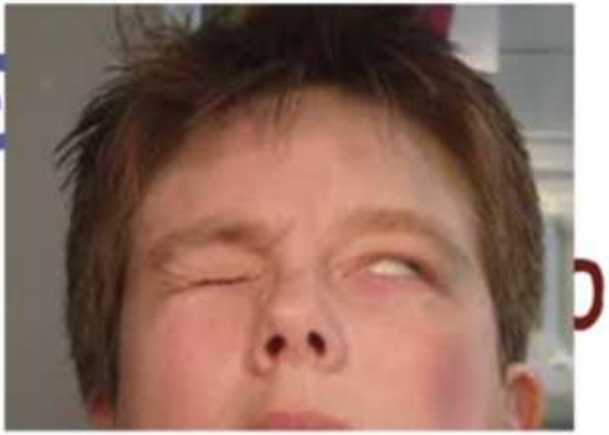
THICKNING OF NERVE

SENSORY

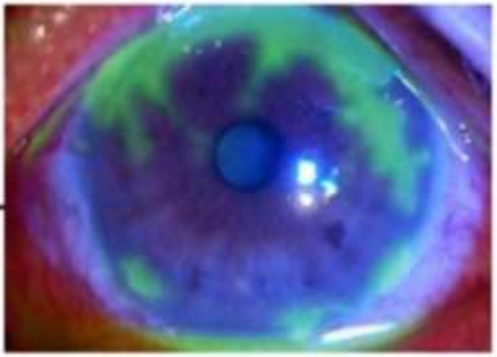
MOTOR



NE



F NERVE



OPTHALMOLOGIC

FACIAL NERVE CLOSER OF EYE LID

TRIGEMINAL NERVE LOSS OF SENSATION CORNEAL ULCER AND TRAUMA

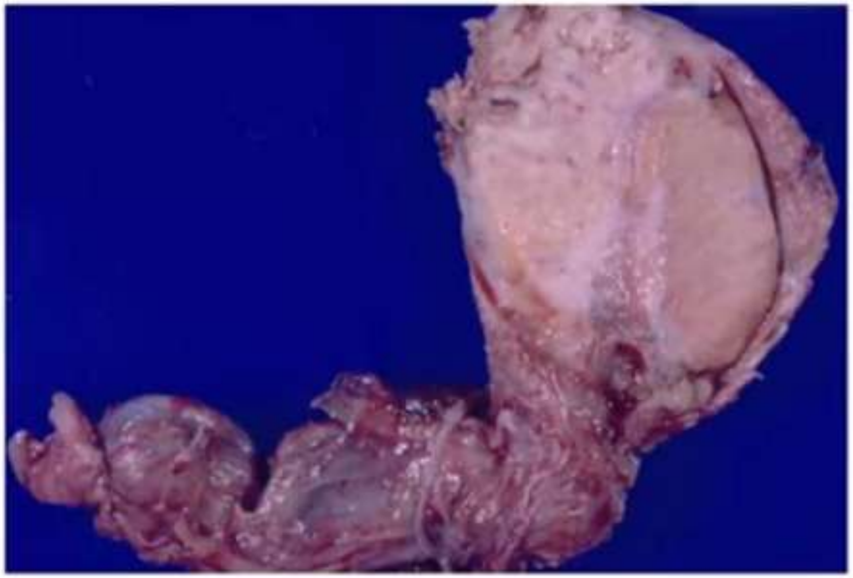
CORNEA AND CONJUNCTIVA

LEPROMATOUS LEPROSY

MULTI ORGAN INVOLVEMENT

TESTIS

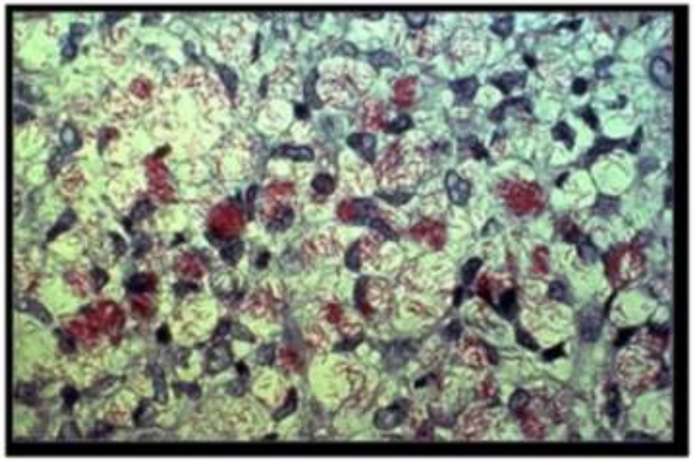
ORCHITIS



DIAGNOSIS

ACID FAST STANING

SPLIT SKIN SMEAR



DIAGNOSIS

ACID FAST STANING

NO BACTERIA TUBERCULOID LEPRASY

NEGATIVE RESULT FOR TUBERCULOID

SPLIT SKIN SMEAR

SKIN BIOPSY

SEROLOGY

PCR DNA

TREATMENT

MULTI DRUG THERAPY

TUBERCULOID

RIFAMPICIN

BACTERICIDAL

ORANGE COLOR IN BODY FLUID

LEPROMATOUS

RIFAMPICIN

TREATMENT

MULTI DRUG THERAPY

TUBERCULOID

RIFAMPICIN

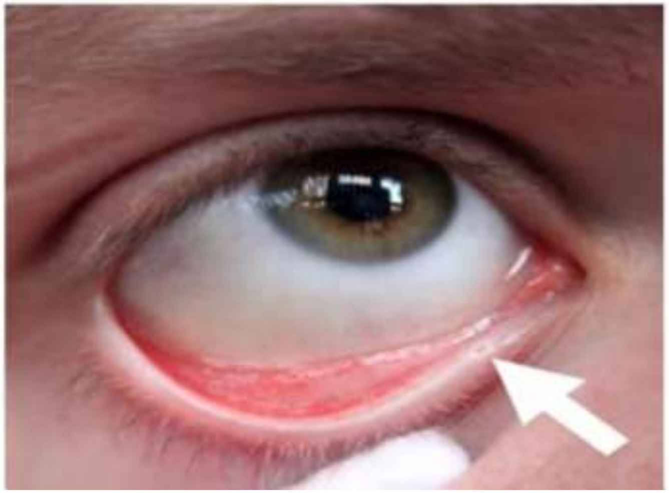
- BACTERICIDAL
  - ORANGE COLOR IN BODY FLUID

DAPSONE

- BACTERIOSTATIC
  - ANEM

LEPROMATOUS

RIFAMPICIN



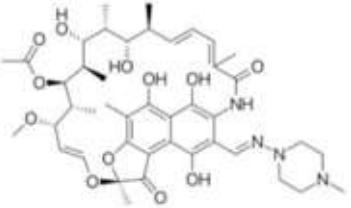
Treatment:

- *Single-lesion Paucibacillary*
  - Single dose of **rifampin**, ofloxacin
- *Paucibacillary*
  - **Dapsone** daily and **rifampin** mont
- *Multibacillary*
  - **Dapsone** daily, **rifampin** monthly and *clofazimine* monthly for 12 months. Additionally, low-dose clofazimine daily for 12 months

Leprosy: Diagnosis & Treatment

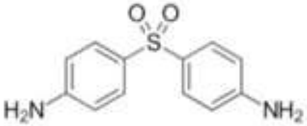
Diagnosis:

- Biopsy of skin lesion
  - *Acid-fast bacilli (AFB) staining*
  - *PCR*
- *Histology*



Treatment:

- *Single-lesion Paucibacillary*
  - Single dose of **rifampin**, ofloxacin and minocycline (ROM)
- *Paucibacillary*
  - **Dapsone** daily and **rifampin** monthly for 6 months
- *Multibacillary*
  - **Dapsone** daily, **rifampin** monthly and *clofazimine* monthly for 12 months. Additionally, low-dose clofazimine daily for 12 months



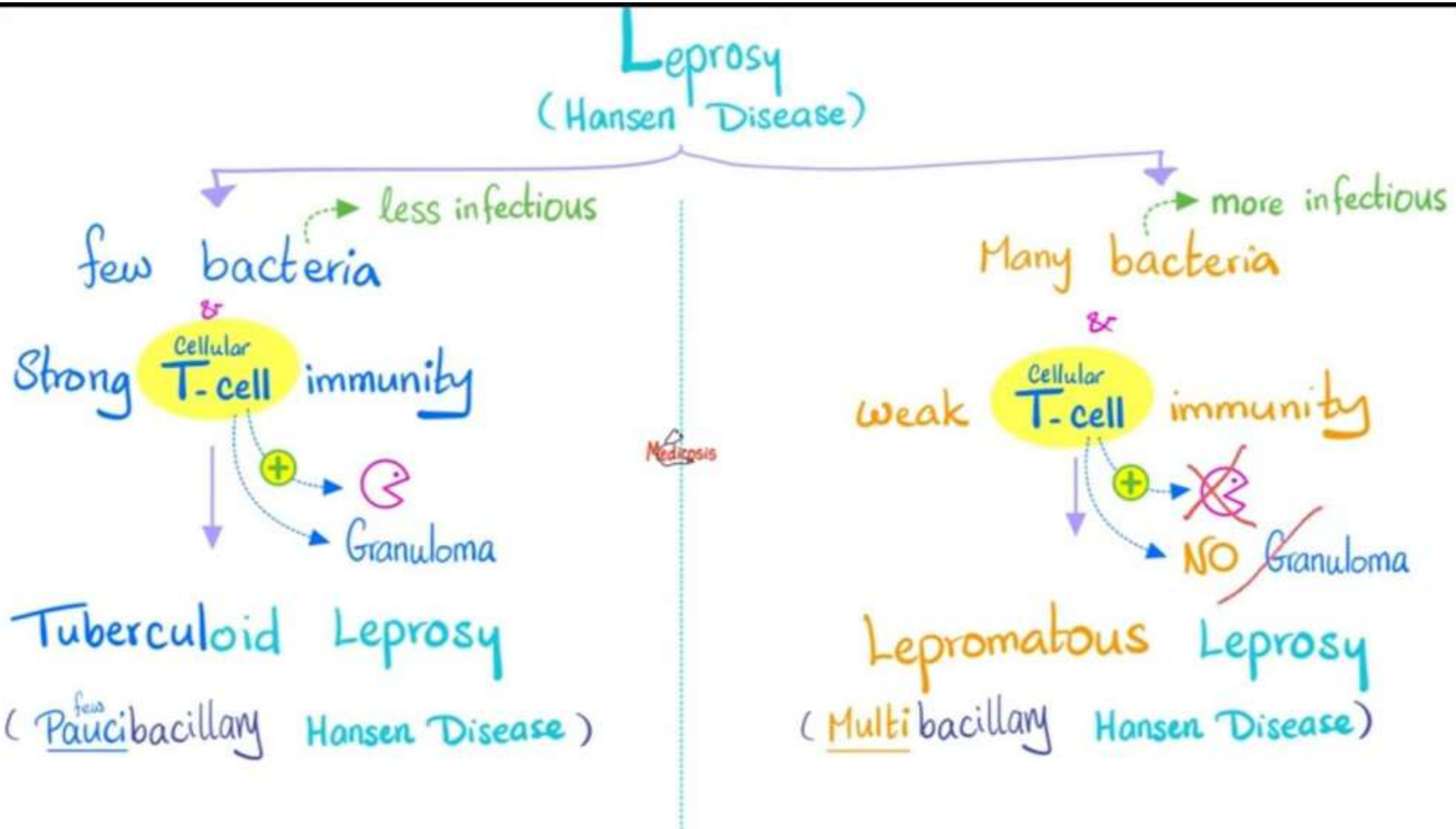
PROGNOSIS

TUBERCULOID

WITHOUT TREATMENT PATIENT SURVIVES

LEPROMATOUS

WITHOUT TREATMENT PATIENT DIE



Leprosy

(Hansen Disease)

few bacteria → less infectious

&

Strong Cellular T-cell immunity

⊕ →

Granuloma

Tuberculoid Leprosy

(few Paucibacillary Hansen Disease)

Many bacteria → more infectious

&

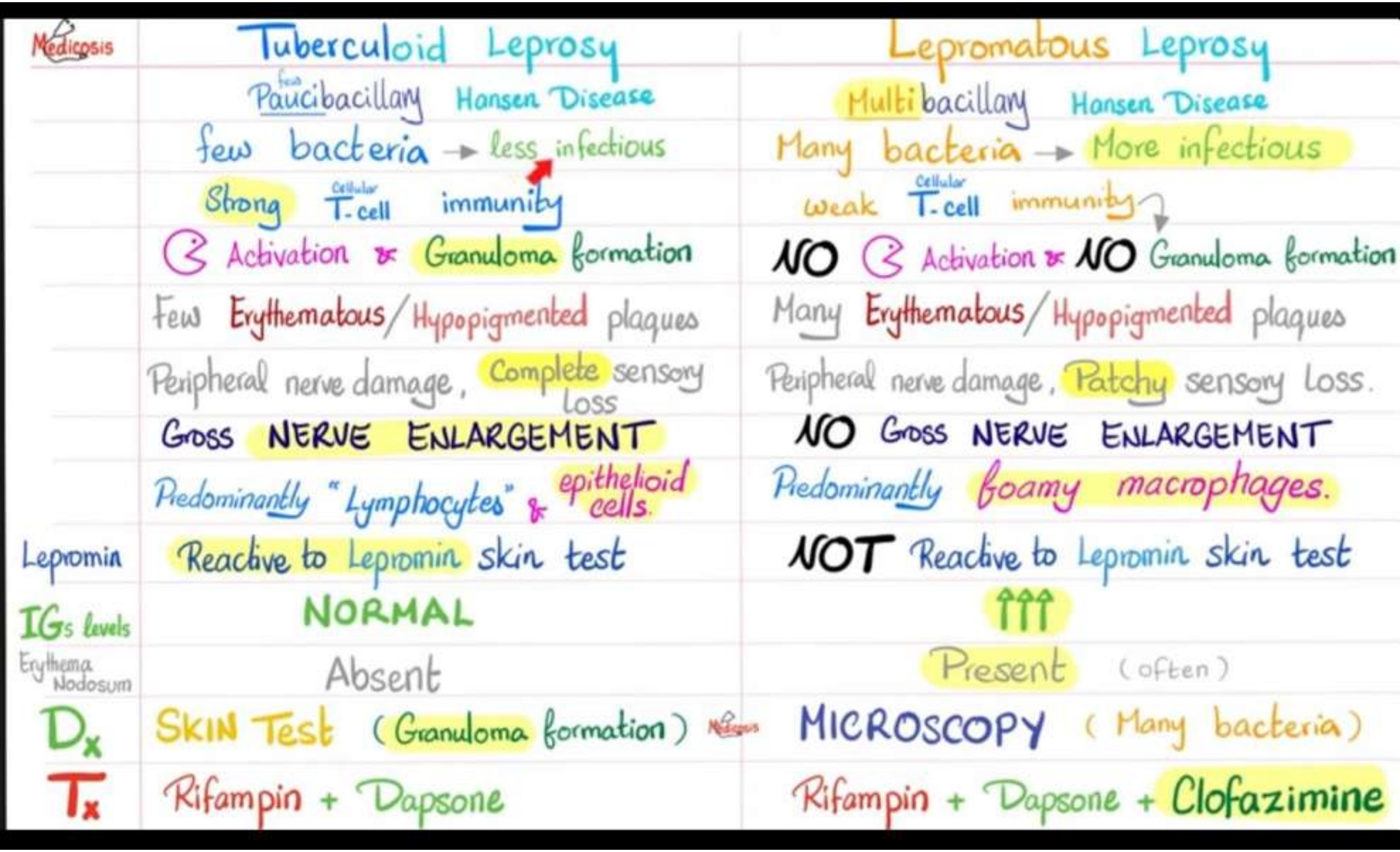
weak Cellular T-cell immunity

⊕ →

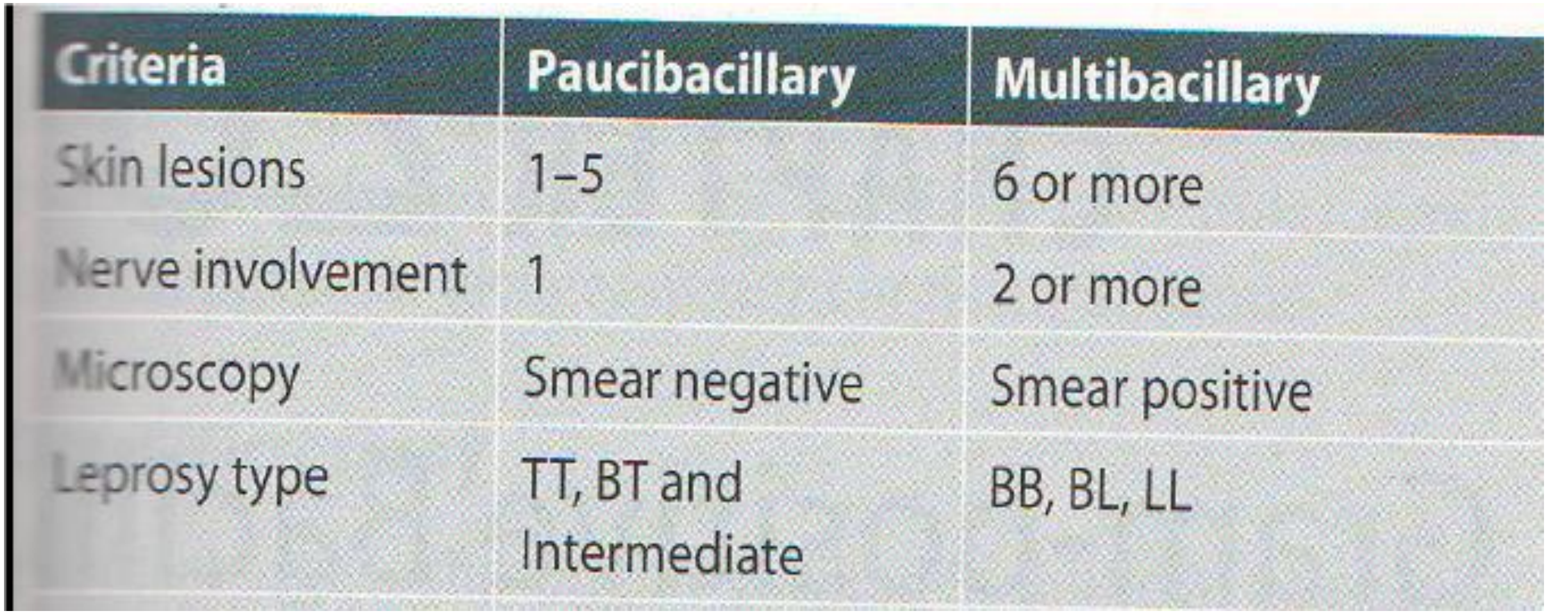
NO ~~Granuloma~~

Lepromatous Leprosy

(Multibacillary Hansen Disease)



	Tuberculoid Leprosy	Lepromatous Leprosy
	$\overset{\text{few}}{\text{Pauci}}$bacillary Hansen Disease	Multibacillary Hansen Disease
	few bacteria → less infectious	Many bacteria → More infectious
	Strong $\overset{\text{Cellular}}{\text{T-cell}}$ immunity	weak $\overset{\text{Cellular}}{\text{T-cell}}$ immunity
	ᗧ Activation & Granuloma formation	NO ᗧ Activation & NO Granuloma formation
	Few Erythematous/Hypopigmented plaques	Many Erythematous/Hypopigmented plaques
	Peripheral nerve damage, Complete sensory loss	Peripheral nerve damage, Patchy sensory loss.
	Gross NERVE ENLARGEMENT	NO Gross NERVE ENLARGEMENT
	Predominantly "Lymphocytes" & epithelioid cells.	Predominantly foamy macrophages.
Lepromin	Reactive to Lepromin skin test	NOT Reactive to Lepromin skin test
IGs levels	NORMAL	↑↑↑
Erythema Nodosum	Absent	Present (often)
D_x	SKIN Test (Granuloma formation)	MICROSCOPY (Many bacteria)
T_x	Rifampin + Dapsone	Rifampin + Dapsone + Clofazimine



Criteria	Paucibacillary	Multibacillary
Skin lesions	1–5	6 or more
Nerve involvement	1	2 or more
Microscopy	Smear negative	Smear positive
Leprosy type	TT, BT and Intermediate	BB, BL, LL

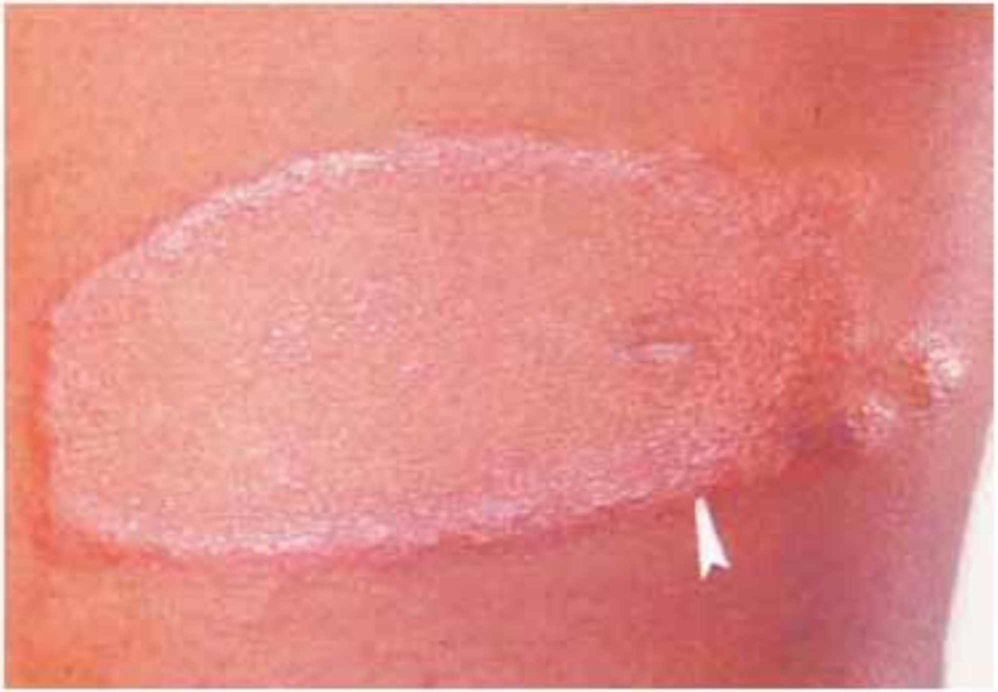


Figure 166-2 **Tuberculoid (TT) leprosy:** a well-defined, hypopigmented, anesthetic macule with anhidrosis and a raised granular margin *(arrowhead)*.

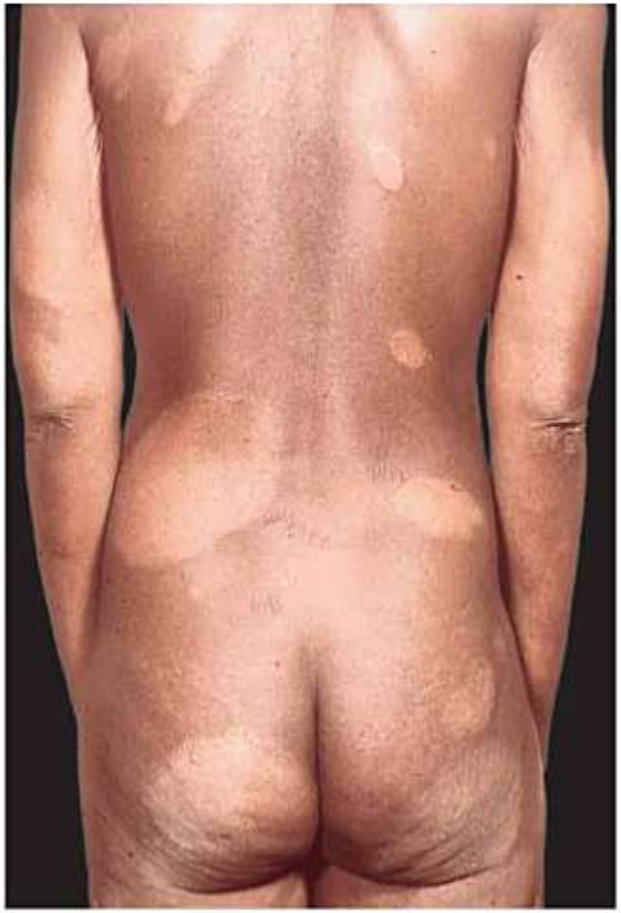
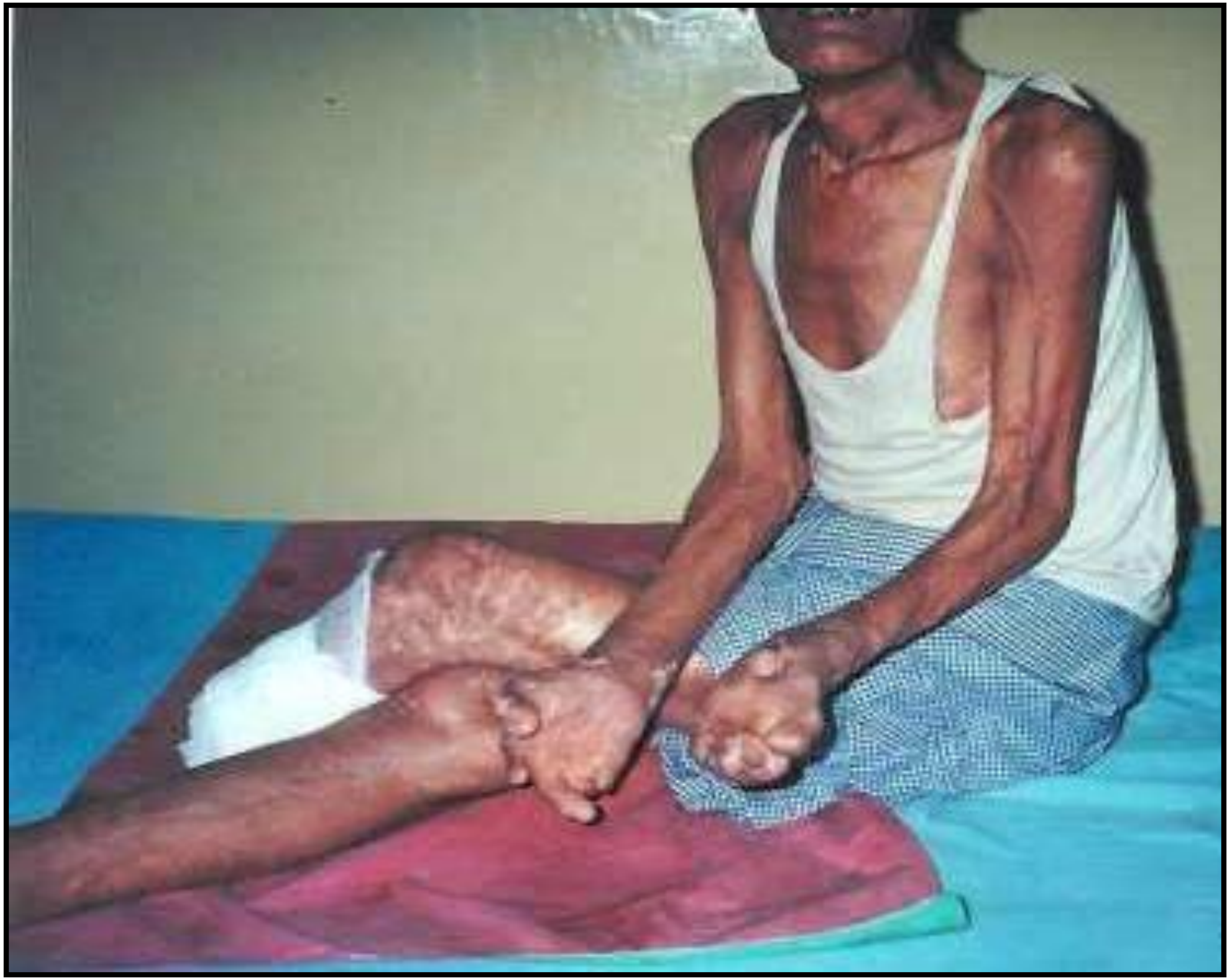
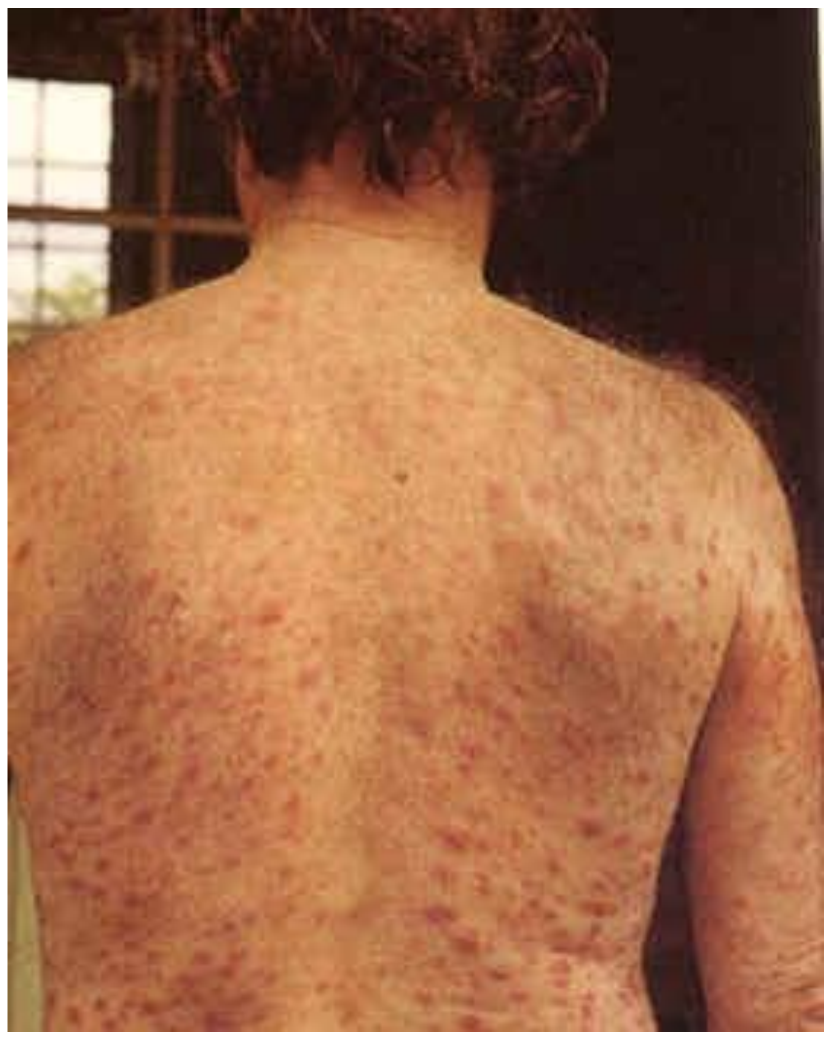


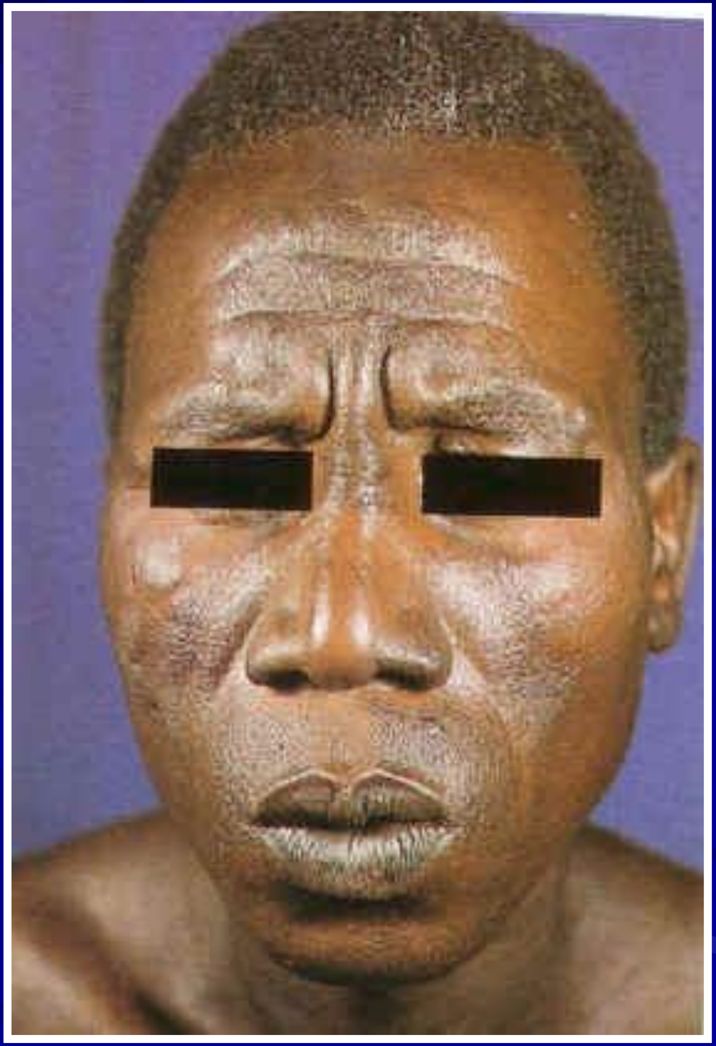
FIGURE 24-60 **Leprosy: tuberculoid type** Well-defined, hypopigmented, slightly scaling, anesthetic macules and plaques on the posterior trunk.



Lepromatous leprosy



Lepromatous leprosy



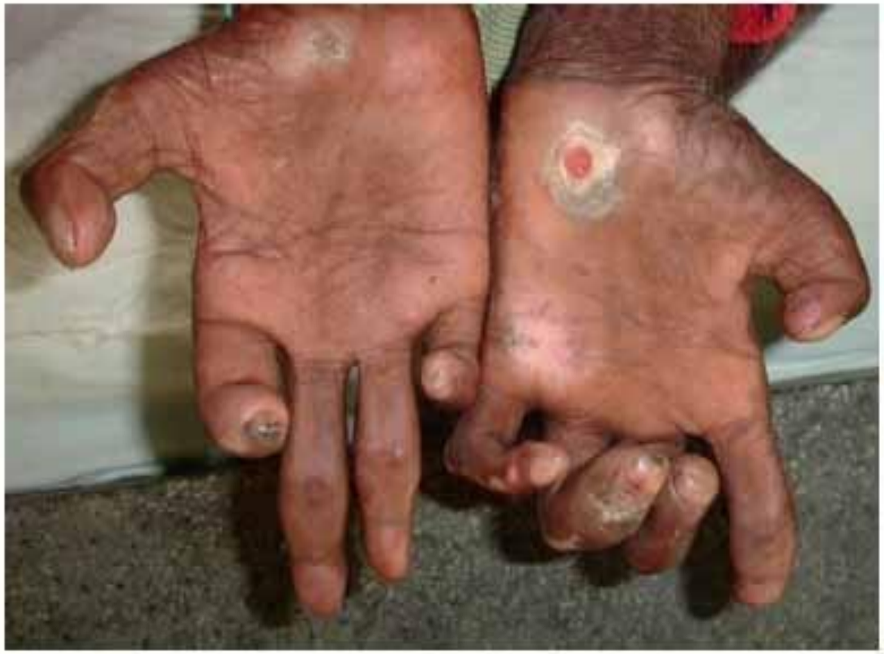


Figure 41.9 Typical leprosy hand deformities: ulnar claw, 'ape thumb' and pressure ulceration.

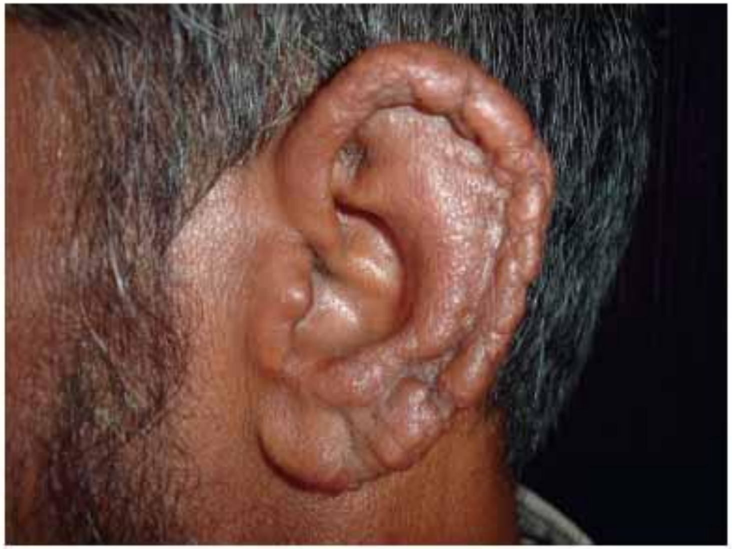


Figure 41.8 Nodular changes in ear lobe in lepromatous leprosy.

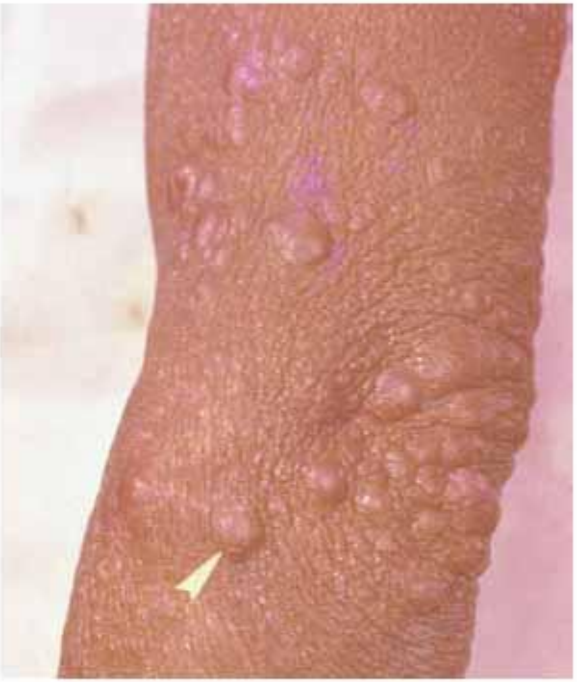


Figure 166-3 **Lepromatous (LL) leprosy:** advanced nodular lesions.

THANK YOU

PYDERMA & BACTERIAL INFECTIONS

Prepared by :

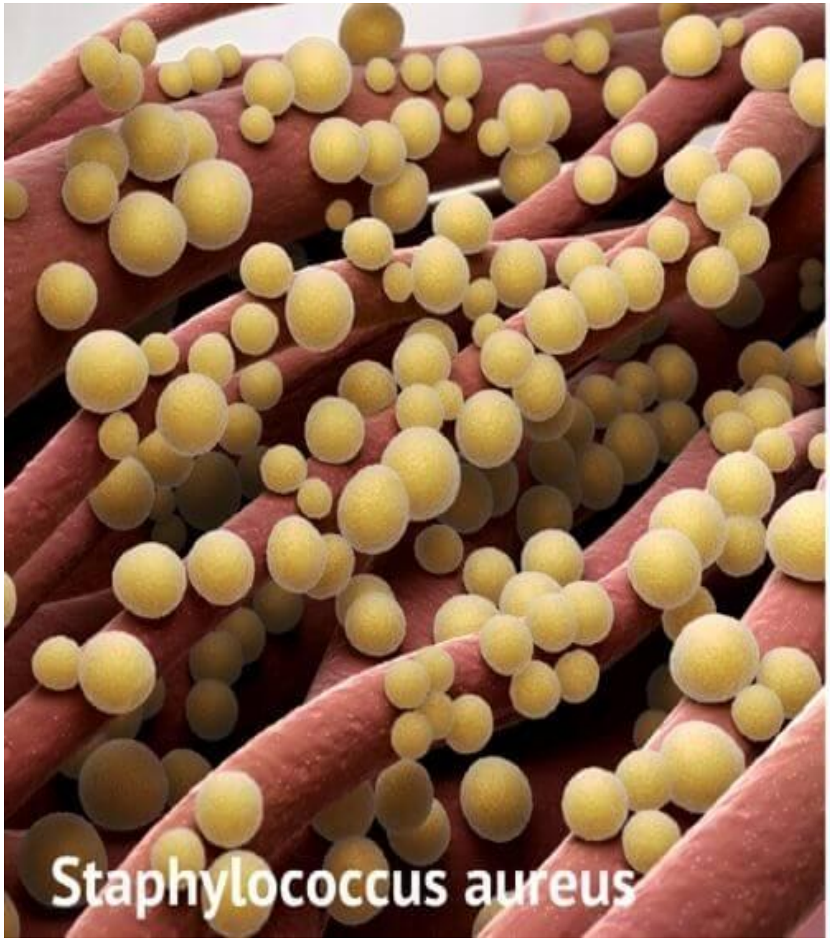
DR.Niveen Almaisary

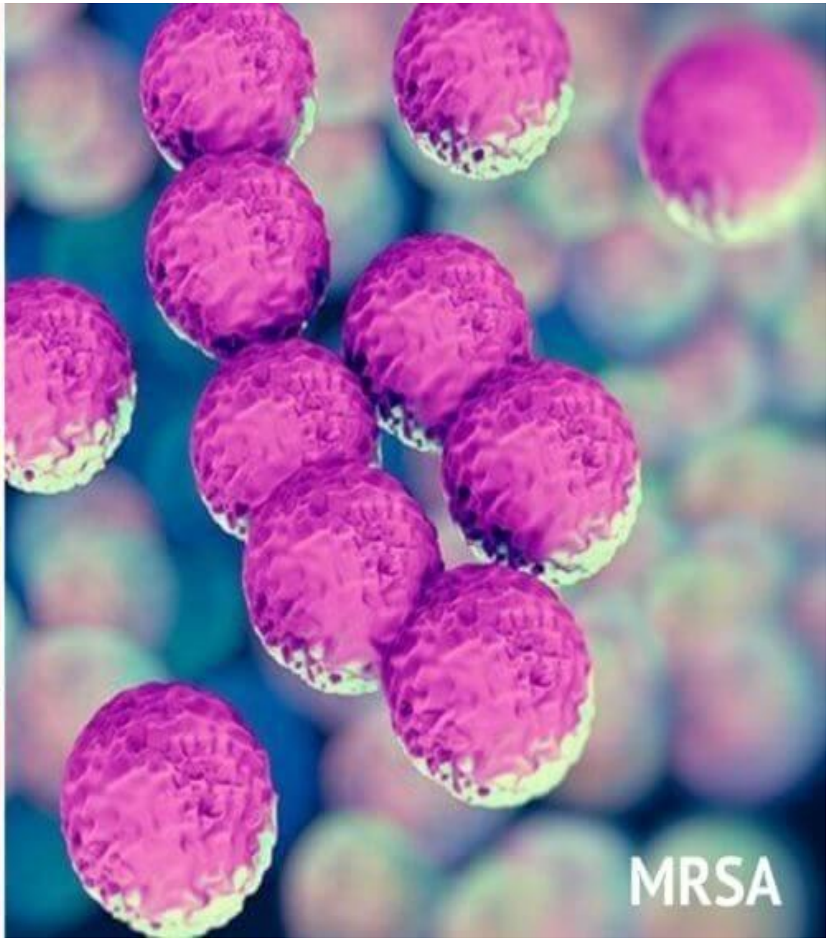
PYODERMA

- means “pus in the skin
- any condition that results in the accumulation of neutrophilic exudate can be termed a pyoderma
- Most commonly, however, pyoderma refers to bacterial infections of the skin
- Pyodermas are caused mostly by **Staphylococcus aureus** and **Streptococcus** pyogenes
- . Most pyodermas are more frequent during the summer and rainy seasons.

STAPHYLOCOCCUS AUREUS

- The Gram-positive cocci bacterium Staphylococcus aureus is the main pathogenic species that causes skin infection).
- Up to 10–20% of the general population are persistent carriers of S. aureus
- Skin infections with S. aureus are more common in patients with breaks in the skin such as scratches, wounds, and ulcers,


Staphylococcus aureus


MRSA

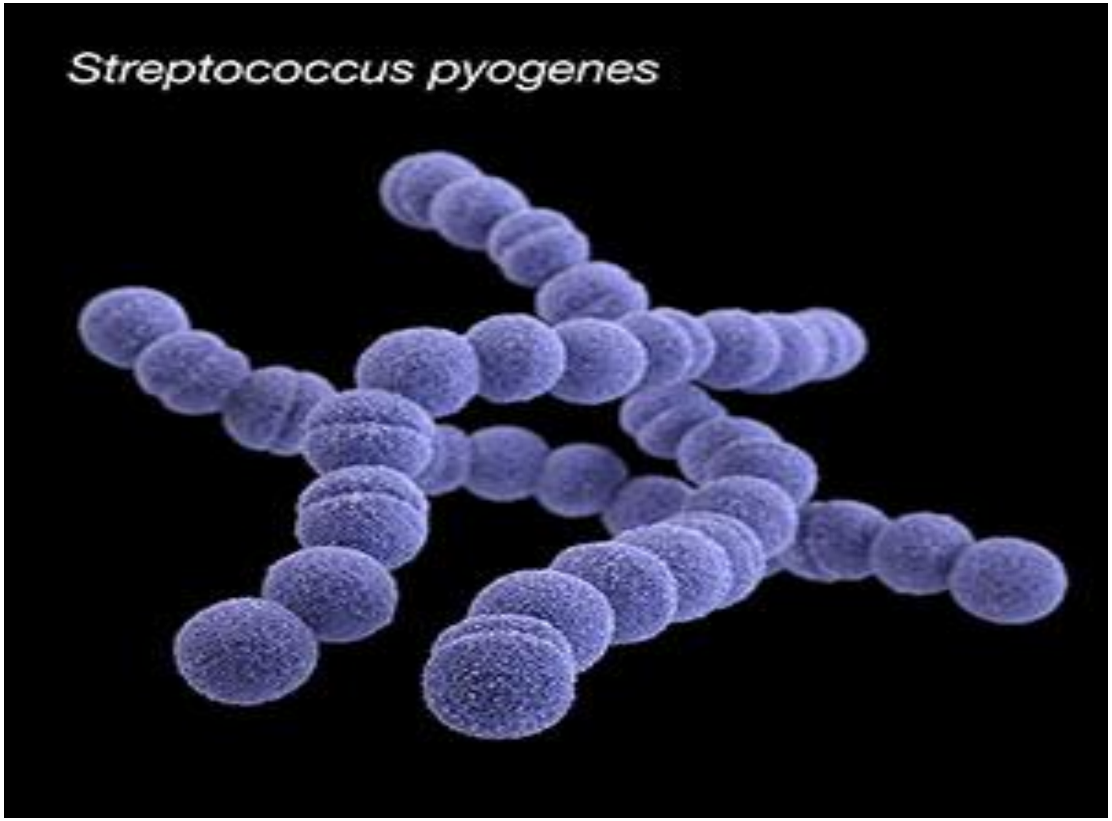
INVOLVEMENT OF S. AUREUS IN CUTANEOUS DISEASE

- **Direct infection of skin and adjacent tissues**
- Impetigo
- Ecthyma
- Folliculitis
- Furunculosis
- Carbuncle •
- Sycosis •
- Occasionally in cellulitis
- **Others Secondary infection :**
- Eczema, infestations, ulcers, etc

- **Cutaneous disease due to effect of bacterial toxin**:
- Staphylococcal scalded skin syndrome
- Toxic shock syndrome • Staphylococcal scarlatina
- Recurrent toxin-mediated perineal erythema

STREPTOCOCCI

- Gram-positive catalase-negative cocci, which are nearly all facultative anaerobes. Streptococcal bacteria are classified into group α, β or γ according to their ability to haemolyse red blood cells:
- The major streptococcal pathogens in humans belong to **group A streptococcus** (GAS), collectively referred to as Streptococcus pyogenes.


Streptococcus pyogenes

STREPTOCOCCI

Direct infections of skin or subcutaneous tissue

- impetigo
- • Ecthyma
- Erysipelas
- Cellulitis
- Vulvovaginitis
- Perianal infection
- Streptococcal ulcers
- Blistering distal dactylitis Necrotising fasciitis
- Others

Tissue damage from circulating toxin

- Scarlet fever
- Toxic-shock-like syndrome
- Recurrent toxin-mediated perineal erythema

INFECTION IS USUALLY PREDISPOSED BY DIFFERENT FACTORS MAINLY:

- General diseases lowering the general defense mechanisms.
- Malnutrition
- Mild trauma to skin
- Insect bites
- Abraded skin lesions
- Poor hygiene
- Impetigo contagiosa may complicate certain skin diseases such as pediculosis, scabies and eczematous eruptions.

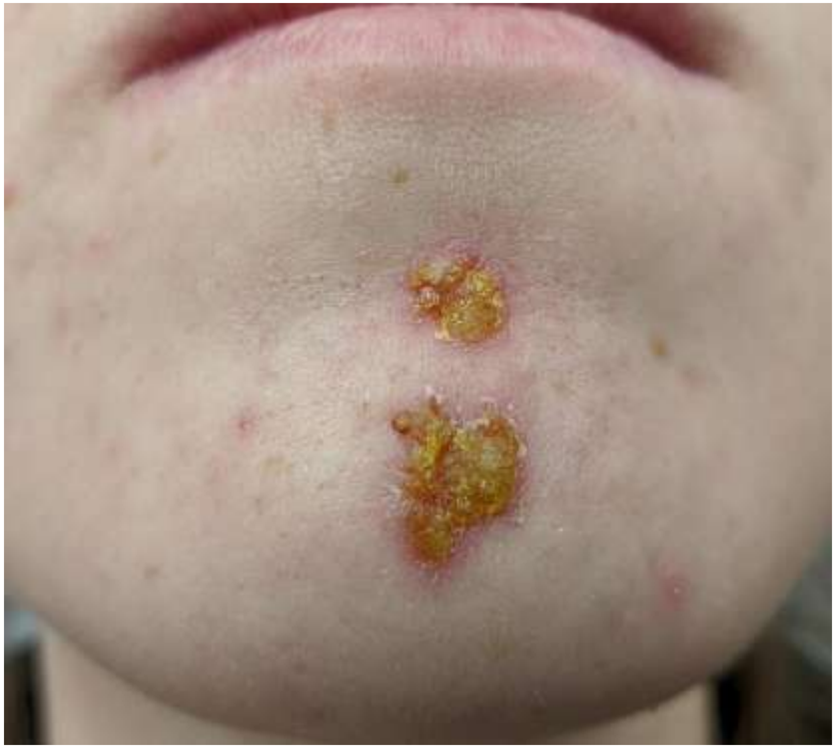
BACTERIAL INFECTION OF SKIN LAB. DIAGNOSIS

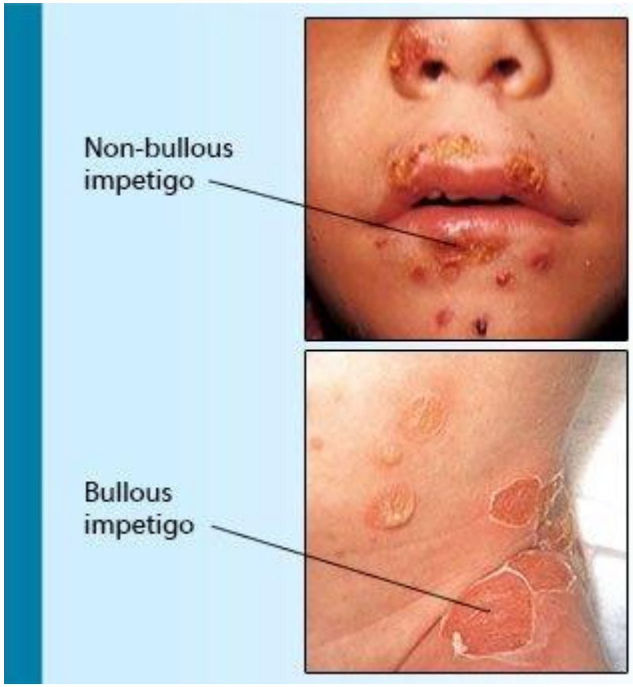
- ☐ Specimen collection.
- ☐ Skin biopsy
- ☐ Skin swab
- ☐ Pus swab
- ☐ Nasal / skin swab

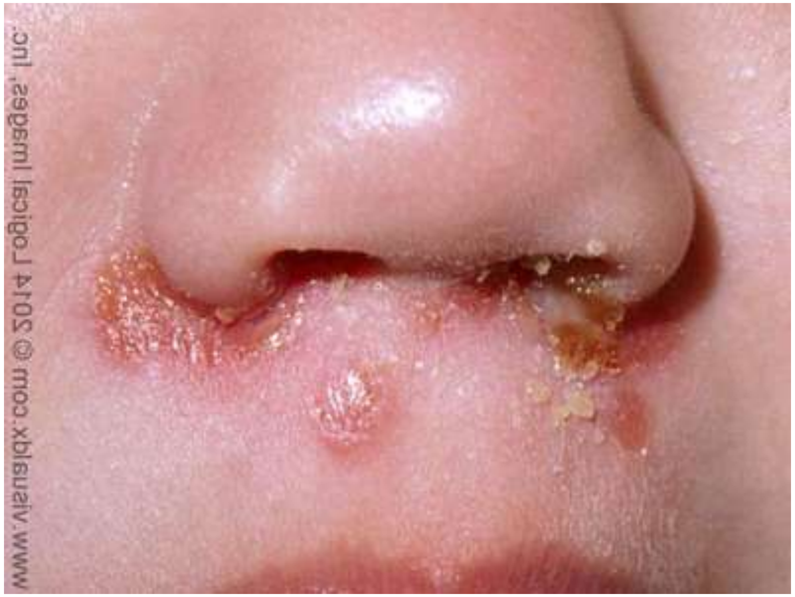
IMPETIGO CONTAGIOSA

- The most common skin infection in children.
- Impetigo is a **contagious superficial bacterial skin infection caused by staphylococci and to a lesser extent by streptococci pyogenes**.
- Either bullous or non bullous
- Initially a vesicular infection that rapidly evolves into pustules that ruputrue, with dried discharage forming **honey-colored crust** on an erythematous base

IMPETIGO




Non-bullous impetigo
Bullous impetigo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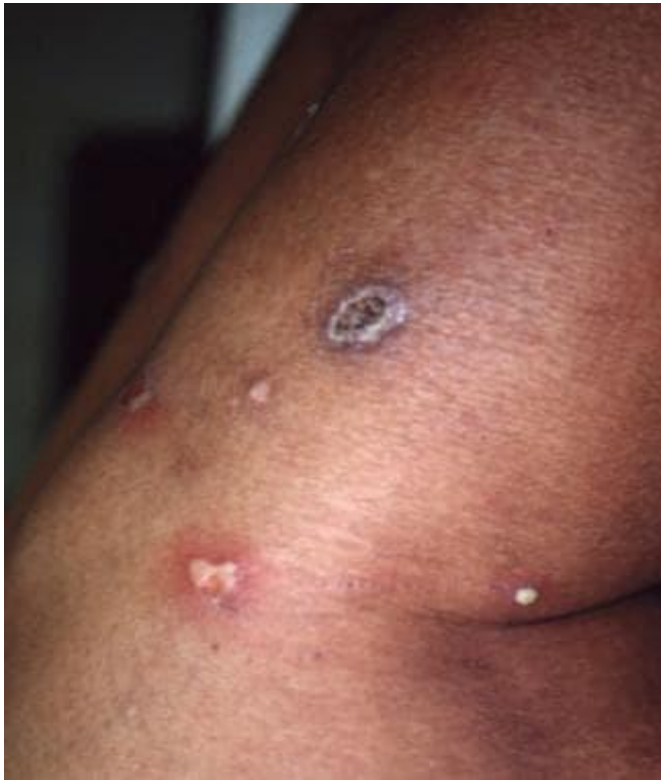
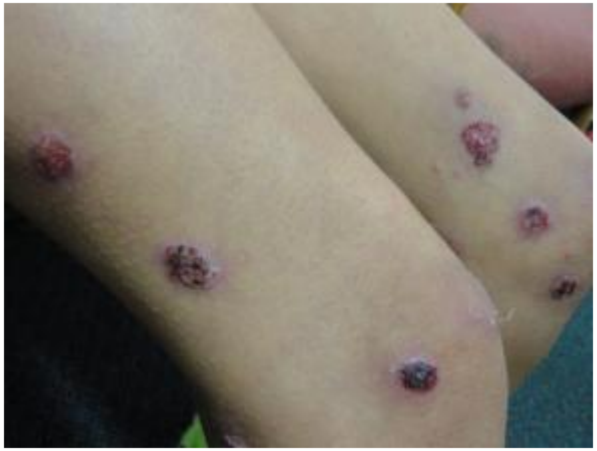
- **Sites** : exposed parts eg,face,extermities,scalp .
- The lesions have little or no surrounding erythema. Itching and scratching leads to autoinoculation on any body surface.
- Most cases heal without scar mainly.
- **Complications:**
- Post –streptococcal acute GN

ECTHYMA

- Ecthyma has many feartures similar to impetigo; (ulcerative impetigo)
- The lesions begins as vesicles or vesiculopustules with erythematous base.
- The process extends more deeply penetrating through the epidermis to produce a shallow ulcer.
- occurs most commonly on the legs & thighs
- - heal with scarring

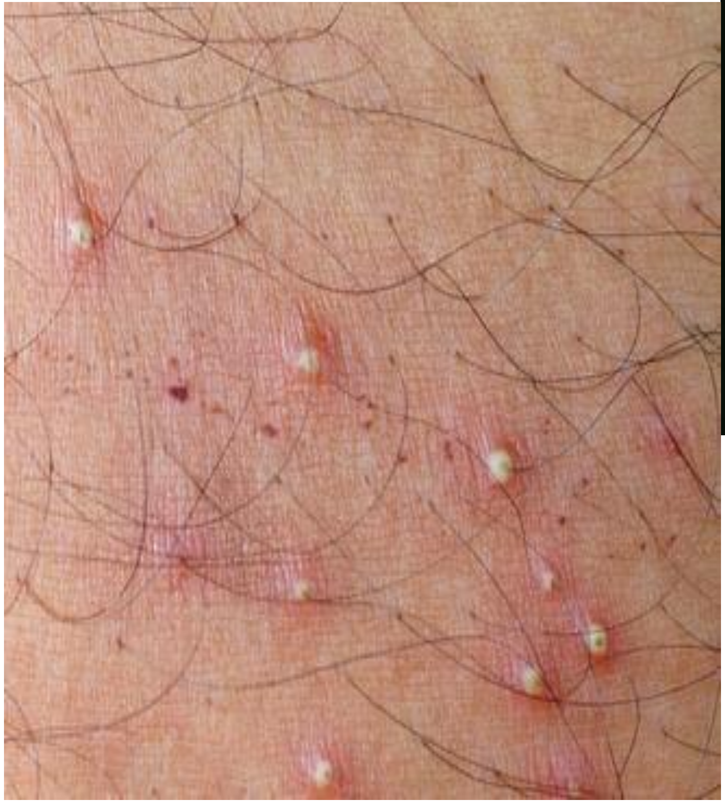
ECTHYMA

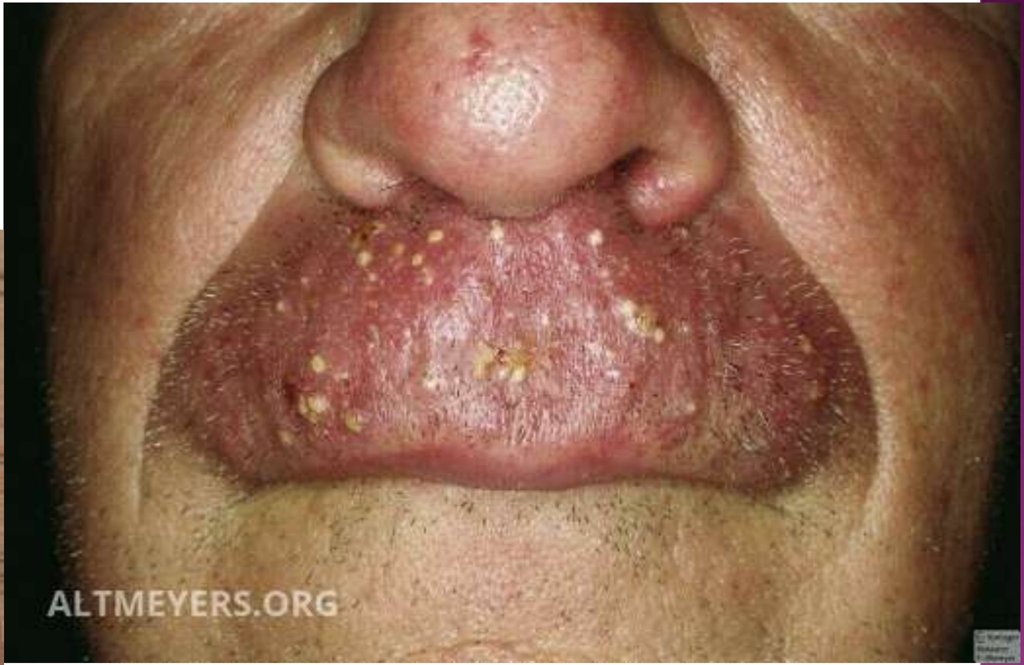


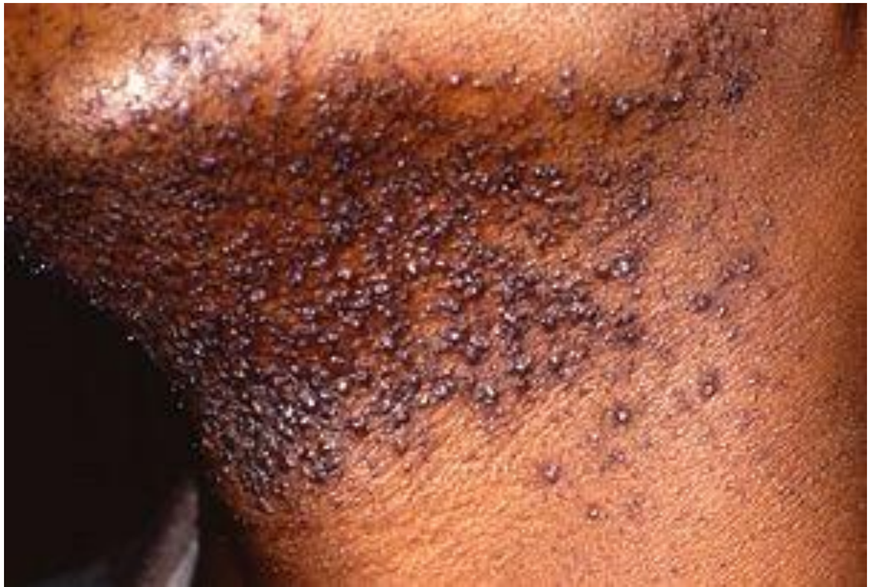


FOLLICULITIS

- Inflammatory diseases of the hair follicles opening which may be infectious or non infectious The lesions may range from tiny white-topped pustules to large, yellow pus-filled lesions.
- may lead to furunculosis, and if left untreated, it may lead to cellulitis (a more extensive inflammation of the skin).
- Absces formation is the major complication of bacterial folliculitis.
- **Therapy**: topical antibiotics and/or oral antibiotics




ALTMEYERS.ORG



CLINICAL VARIENT

Superfacial folliculitis ,bockharts impetigo

- Adome –shaped pustule at the orifice of a hair follicle that heals within 7-10 days
- Caused by staph.aureus and effects mainly extremities &scalp
- Topical steroid are acommon predisposing factor.

Sycosis vulgaris

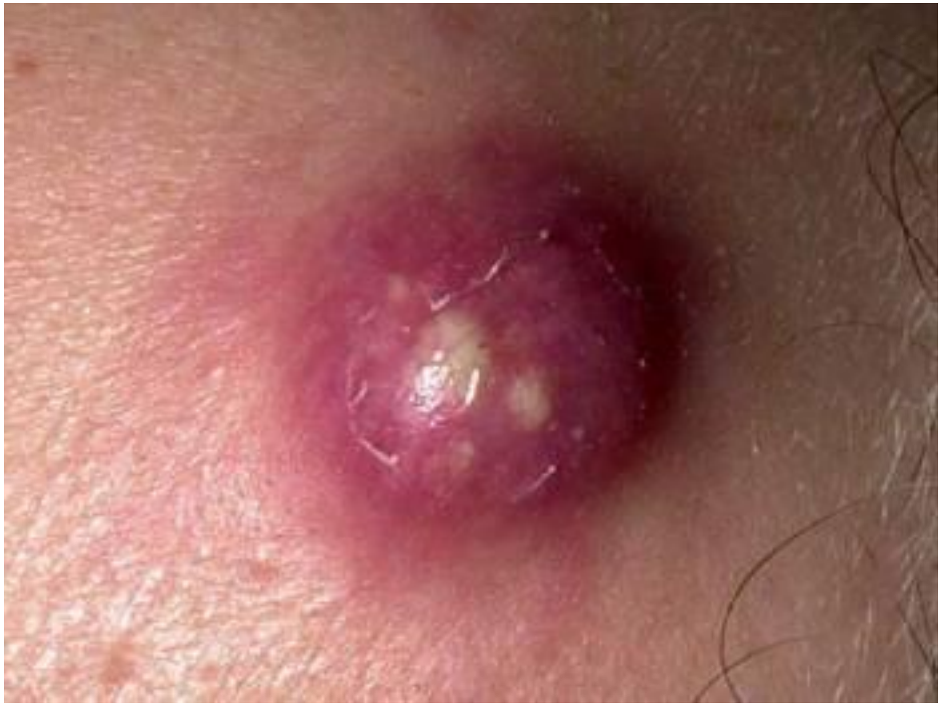
- Recurrent red follicular papules or pustules on the hair ,usually remain discrete over the beard or upper lip,but may coalesce to produce rasied plaques studded with pustules

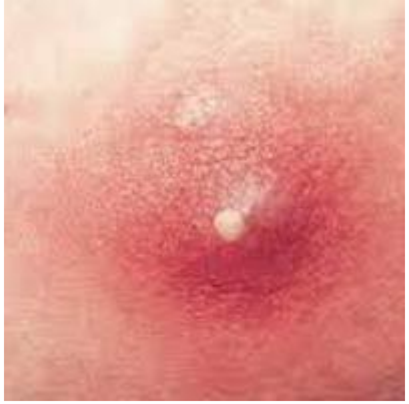
pseduofolliculitis

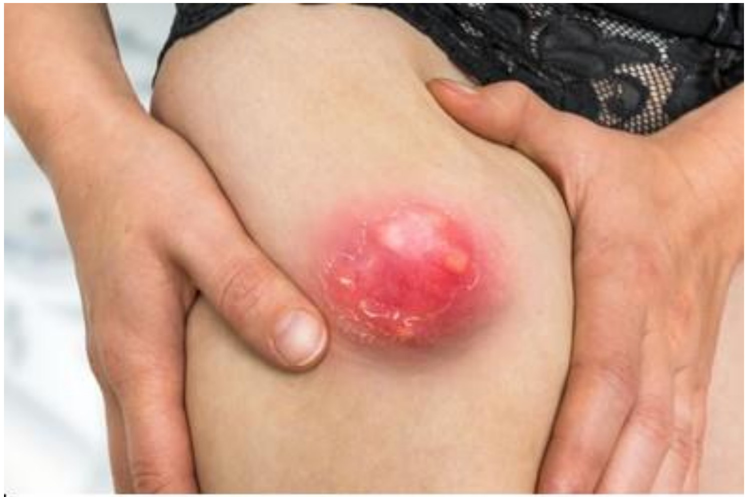
result from penetration into the skin of skin of sharp tips of shaved hairs

- **A furuncle**
  - is an acute, round, firm, tender, circumscribed, perifollicular staphylococcal pyoderma that usually ends in central suppuration.
- **A carbuncle**
  - is two or more confluent furuncles with separate heads. Some lesions disappear before rupture, but most undergo central necrosis and rupture through the skin, discharging firm, purulent, necrotic debris.
  - Common sites include the nape, axilla, and buttocks, but lesions may occur anywhere.

- **Treatment** consists of warm compresses and oral antibiotics. Surgical drainage may be required







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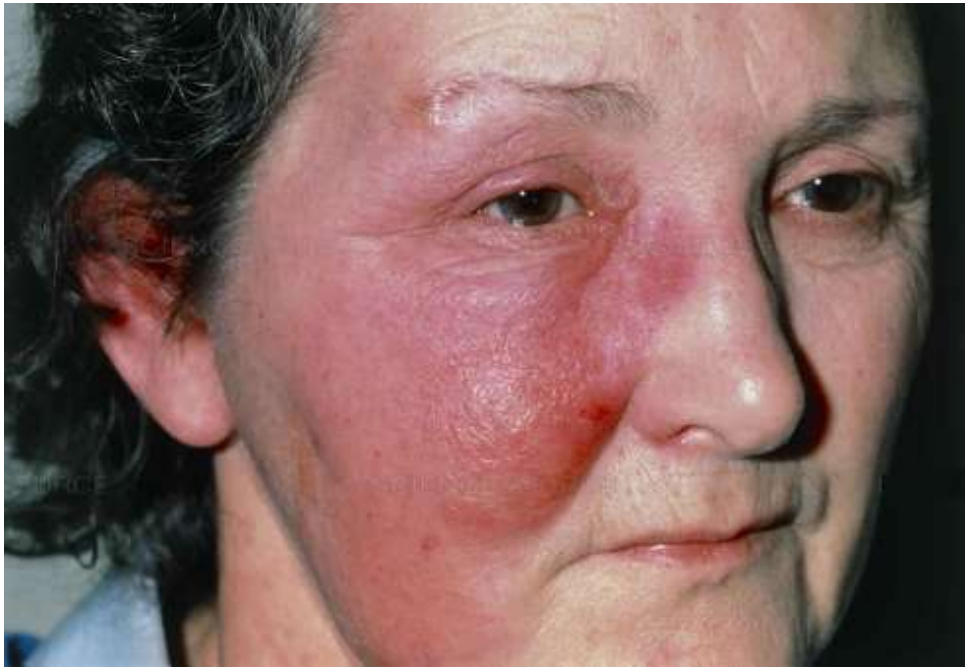
ERYSIPELAS

- is an acute beta-hemolytic group A streptococcal infection of the skin involving the superficial dermal lymphatics that causes marked swelling.
- - is characterized by local redness, heat, swelling, and a highly characteristic raised, well-defined border The legs and face are the most frequently affected sites.
- The onset can be preceded by prodromal symptoms of malaise with or without chills, fever, headache, vomiting, and joint pain.
- When legs are affected, edema and bullous lesions are often present.

ERYSIPELAS



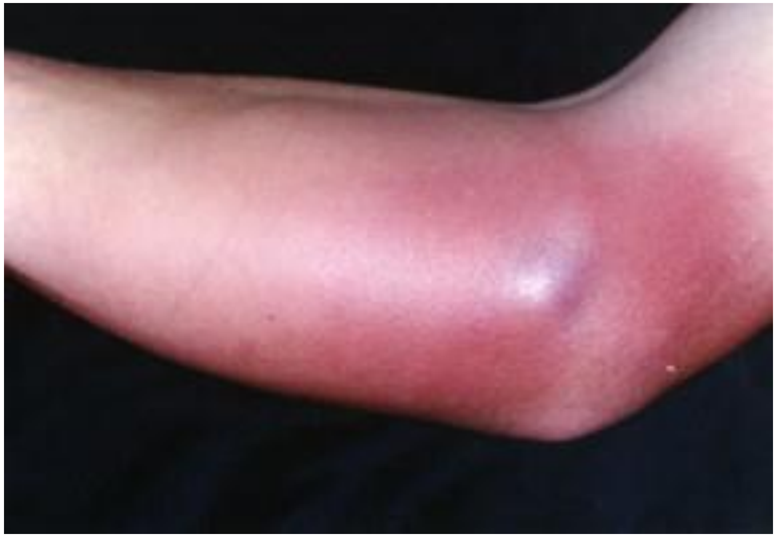
Predisposing medical or postsurgical conditions, such as skin ulcers or eczematous lesions, chronic fungal infections, fissures, local trauma, venous or lymphatic compromise, and obesity are present in many patients who develop erysipelas

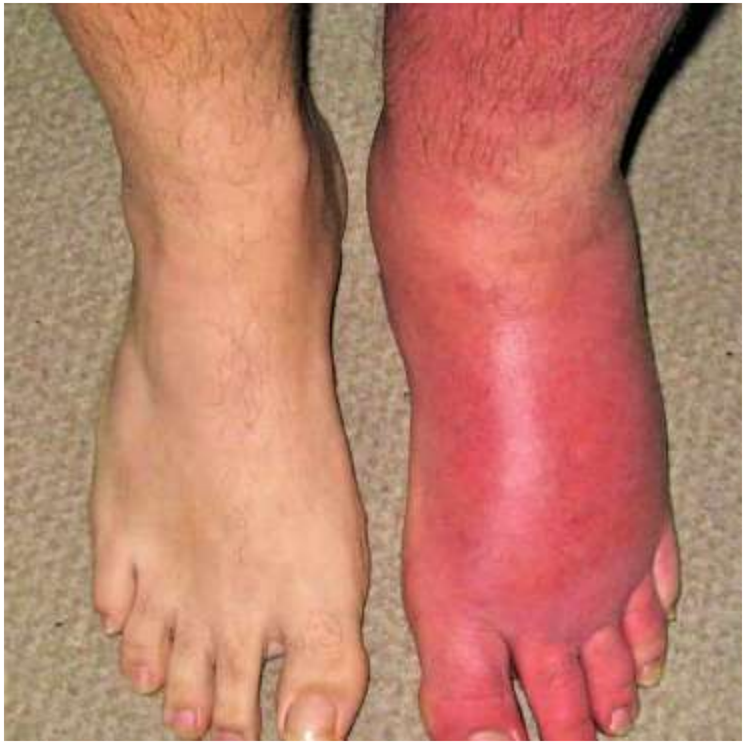
CELLULITIS

- Cellulitis is acute bacterial infection of the skin and subcutaneous tissue most often caused by streptococci or staphylococci
- Symptoms and signs: **pain, rapidly spreading erythema, and edema; fever may occur, and regional lymph nodes may enlarge**.
- Cellulitis infections begin when bacteria enter cracks in the skin (through scrapes, cuts, burns, insect bites, surgical incisions, or intravenous needles

CELLULITIS

- Cellulitis The skin is hot, red, and edematous, often with surface appearance resembling the skin of an orange (peau d'orange).
- The borders are usually indistinct
- Cellulitis is treated with either oral or intravenous antibiotics





Thank
You

VIRAL SKIN INFECTIONS

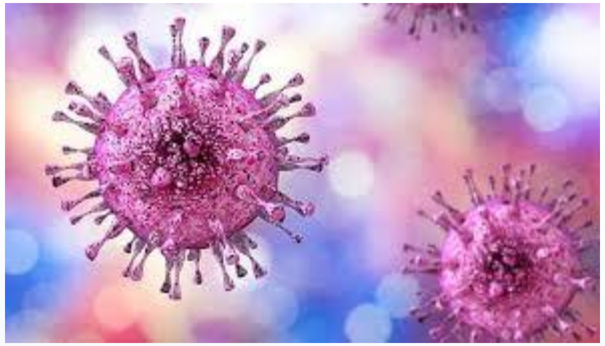
Prepared by :

Niveen Almaisary

The main viral diseases of the skin

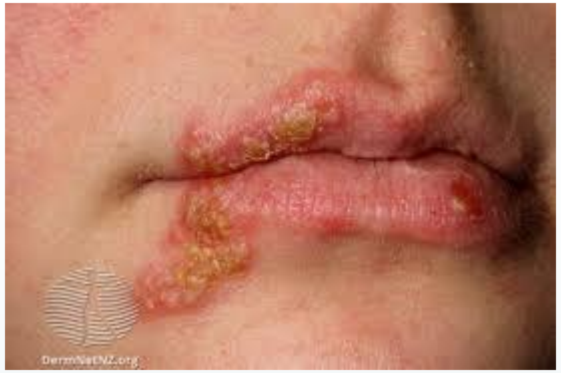
- Herpes simplex
- Chicken pox
- Herpes zoster
- Wart
- Molluscum contagiosum

Herpes simplex



- HSV is a double strand DNA virus
- There are two types of HSV.
- **HSV type 1**,associated with facial or oral infection
- **HSV type 2** , affecting the genitalia
- HSV infection is one of the commonest infections throughout the world .
- HSv1 , infection mainly in children
- HSV2,infection mainly occur after puberty.

Herpes simplex

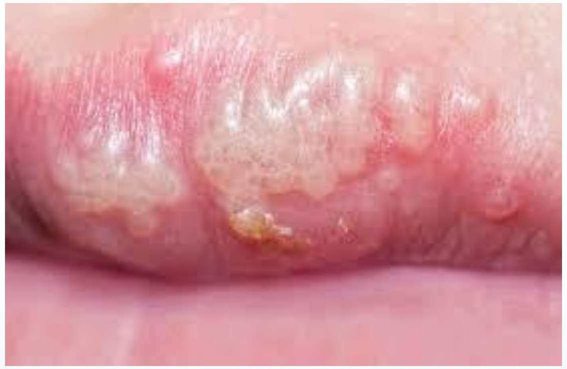


Modes of infection:

Close contact ,Invade mm or abraded skin

- IP: (2_20days)
- Primary attack asymptomatic in 90% of cases
- Later became latent in sensory ganglion

→ Reactivation. → Reccurance of infection



Precipitating factors

- UVR
- menstruation
- Febrile illness
- Common cold
- Stress
- GIT upset
- Immunosuppression

CP :

Primary: Symptomatic in 10%..Children ,Constitutional symptoms….Enlarged LN

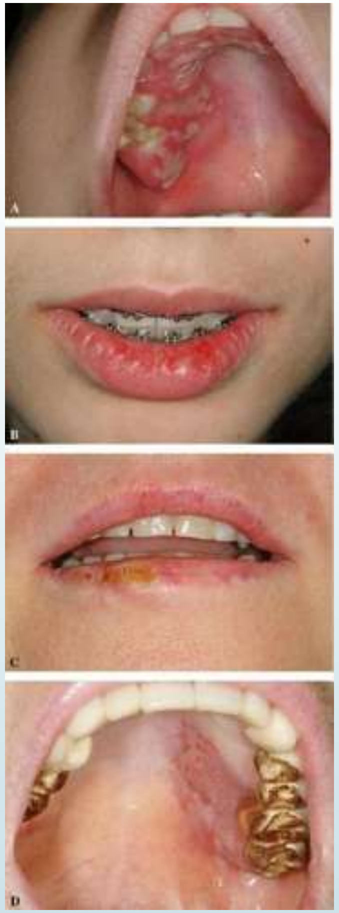
Large vesicles….not grouped……dissiminated

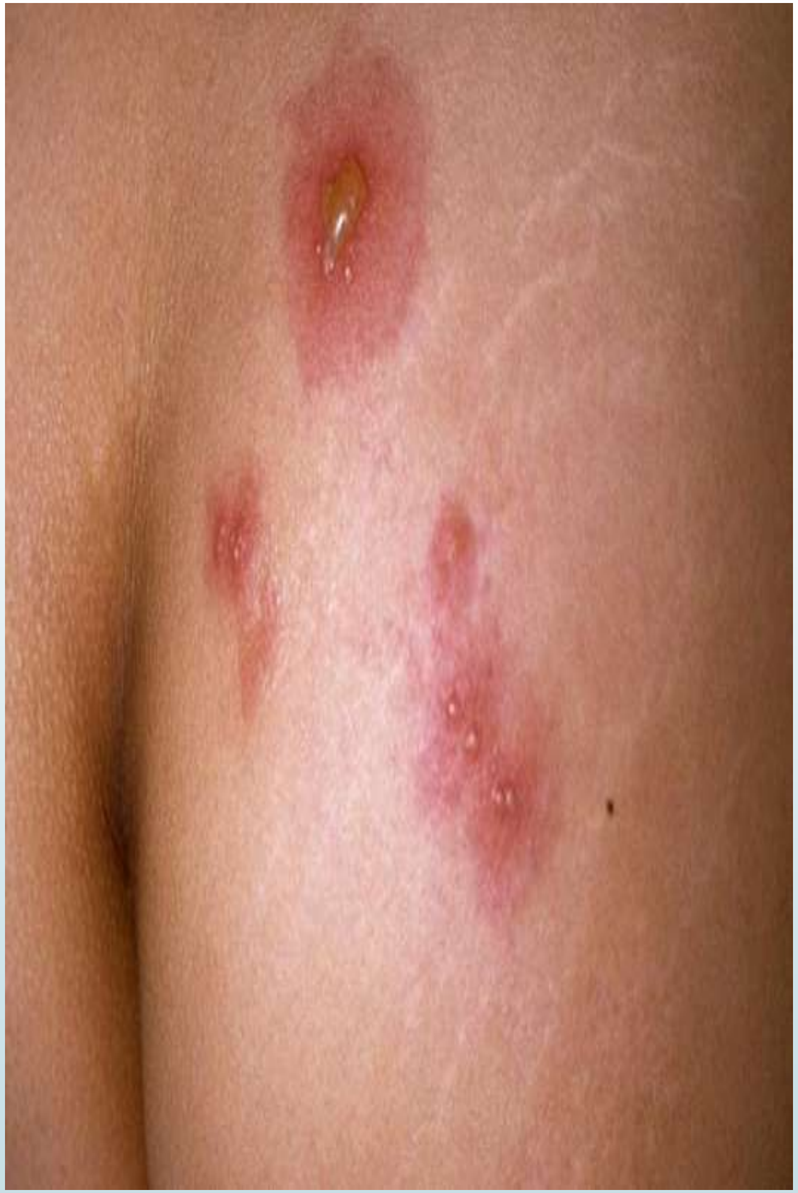
Recurrent

Erythematous patch _ grouped _pustules_crsuty vesicles_PIH

Clinical varient

- Herpctic gingivostomatitis.
- Herpetic labialis
- Herpetic whitlow
- Herpetic keratoconjuctivitis
- Neonatal herpes simplex
- Herpes genitalis
- Eczema herpeticum
- Disseminated herpes simplex

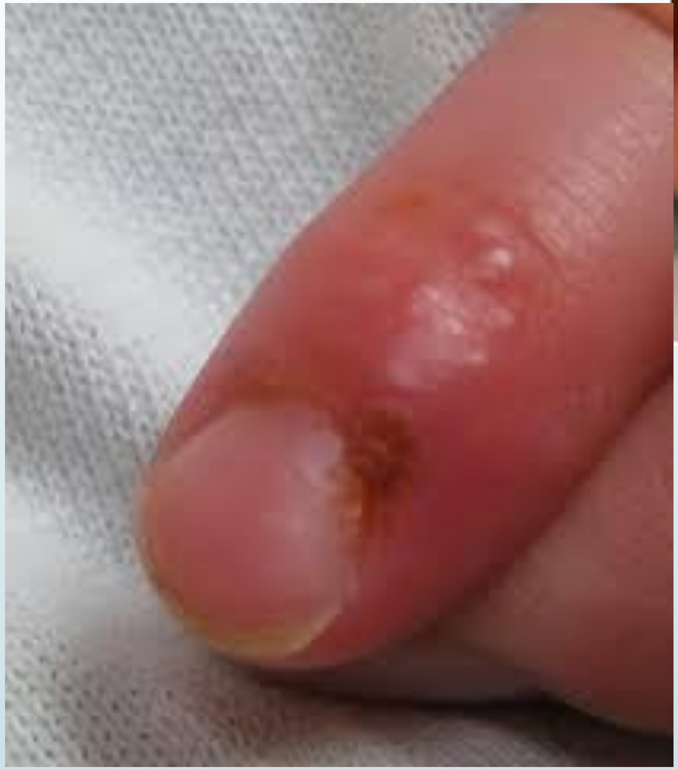


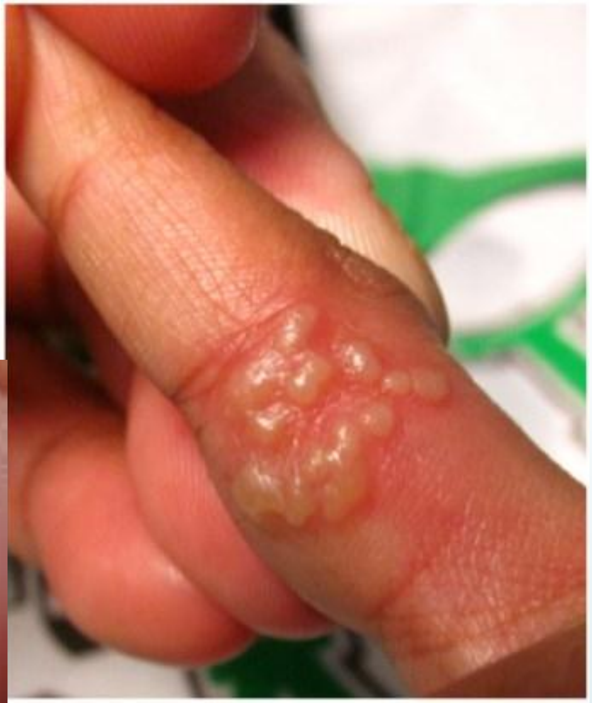


Diagnosis

- Direct microscopic examination
- Tzanck smear
- Viral culture it's the most reliable way to make the diagnosis
- Serology
- PCR

Useful in the diagnosis if culture _ve or atypical lesion



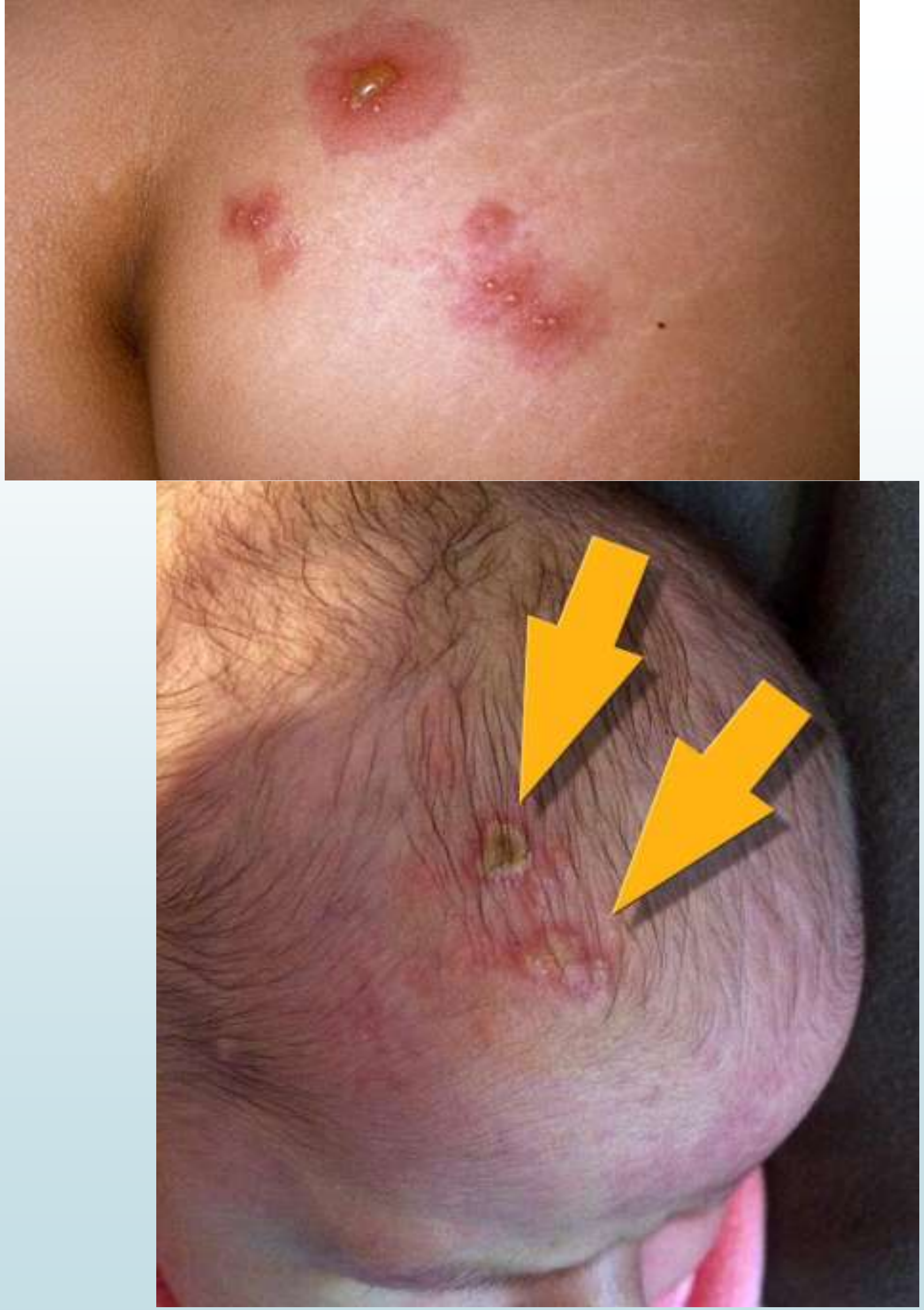


Treatment

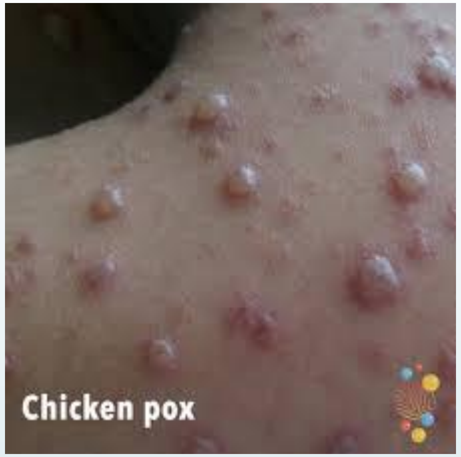
- Most cases is self limited
- Topical: antibiotic, antiseptic lotion, acyclovir cream
- Systemic: Acyclovir, valacyclovir

- Antiviral (acyclovir)

Used to shorten the course of the disease prevent dissemination and recurrence,



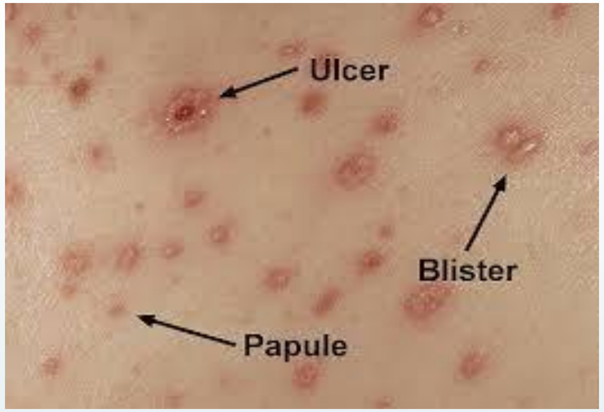
VARICELLA (CHICKEN POX)



- Organism: Varicella zoster (HHV 3)
- IP: 10-20 days
- Mode of infection: Direct contact or droplet
- Usually permanent immunity
- Mainly in children
- Symptomatic in 95% of cases

Clinical picture

- **General**: fever, malaise, headache, enlarged LN
- **Ctanueous:** vesicles – pustules – crustations (polymorphic lesions)
- **Distribution:** Centripetal (mainly on the trunk, face & oral mucosa)



Varicella Complications

- Varicella pneumonia……Fatal
- Neonatal varicella

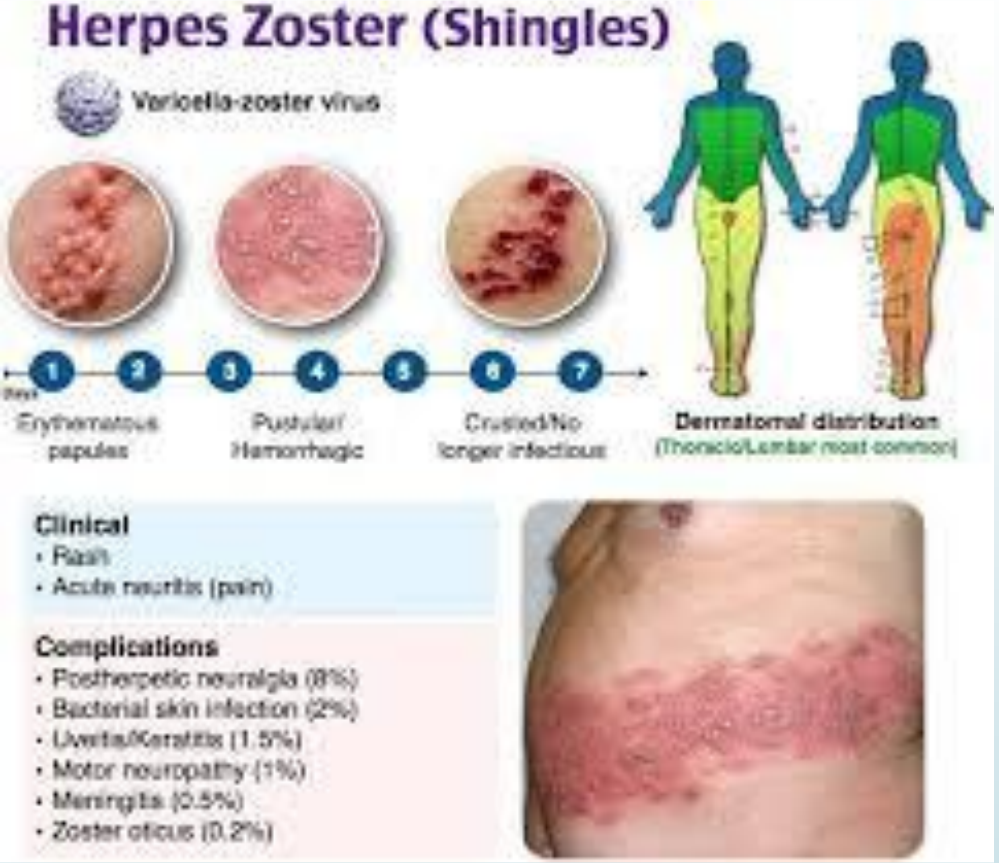
Treatment

- Treatment is symptomatic
- Antipruritic lotions & anti histamines are given for itching
- Antibiotics for secondary infection
- Acyclovir in Severe cases 20mg/kg

HERPS ZOSTER

- Its acute localized viral infection
- characterized by unilateral pain or vesicular rash eruption limited to dermatome innervated by corresponding sensory ganglia.
- **Organism**: Zoster virus (Reactivation of varicella virus)
- **Age group**: Middle aged and elderly
- **Mode of infection**: mostly reactivation of the latent virus in immunosuppressed patients
- **IP**: 2-3 weeks

- **When immunity decrease reactivation to virus occure causing dermatomal pain& skin lesion**

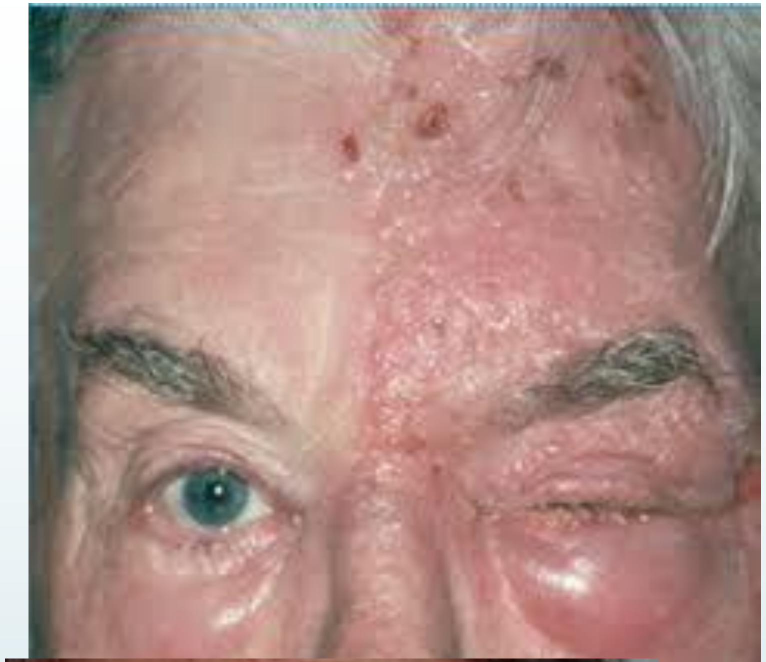


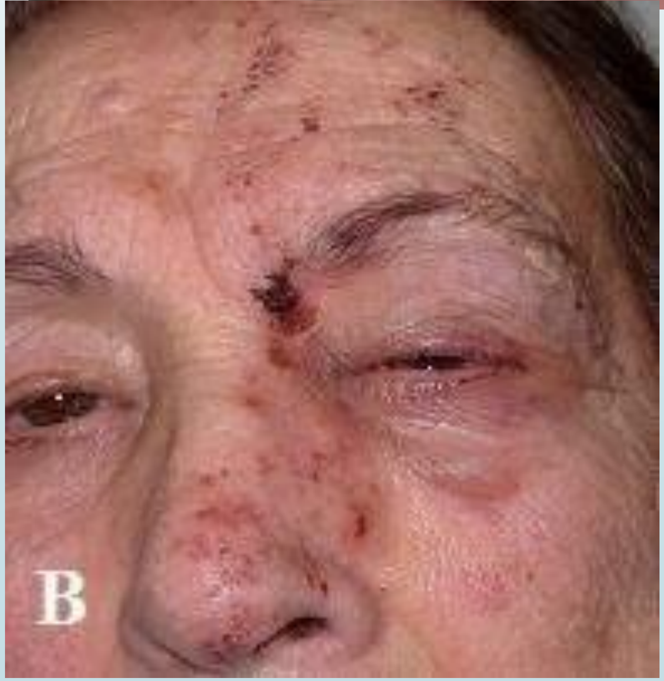
Herpes Zoster (Shingles)

Varicella-zoster virus
1
2
3
4
5
6
7
Days
Erythematous papules
Pustular/ Hemorrhagic
Crusted/No longer infectious
Dermatomal distribution
(Thoracic/Lumbar most common)

Clinical
- Rash
- Acute neuritis (pain)

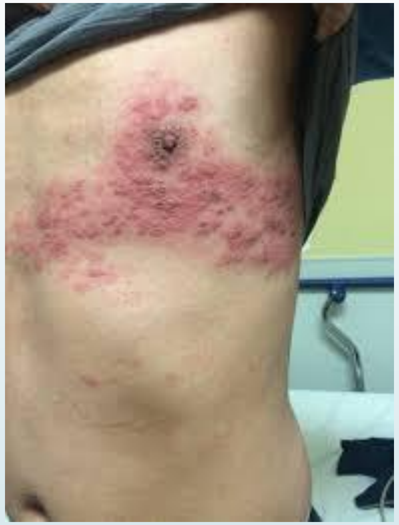
Complications
- Postherpetic neuralgia (8%)
- Bacterial skin infection (2%)
- Uveitis/Keratitis (1.5%)
- Motor neuropathy (1%)
- Meningitis (0.5%)
- Zoster oticus (0.2%)





Clincal Picture

- prodrome of dermatomal pain (3_5) days.
- then closely grouped vesicles & pustules on erythematous base,
- New lesions continue to appear for a week
- heal in 2 –4 weeks with scar
- The regional L .N. are enlarged &tender
- The eruption mostly unilateral and usually affects one dermatome
- Throacic >50 of cases
- Trigeminal region 10_ 20%, Lambosacral & cervical 10_20%



Dx: clinically mainly

- Confirmed by T zanck test
- Viral culture to rule out HSV infection.

Treatment

- The disease is self limited.,the goal of treatment
- To minimize the pain (analgesic)
- Speeding the healing topical &systemic if 2ry bacterial infection
- Prevention of dissemination
- Acyclovir 800/6hr for 7_10 days

Complication

- Post herpetic neuralgia
- Ramsey hunt syndrome
- Disseminated zoster
- Zoster encephalomyleitis
- Ocular complications
- Secondary bacterial infection & gangrene
- Facial palsy

Wart(Verruca)

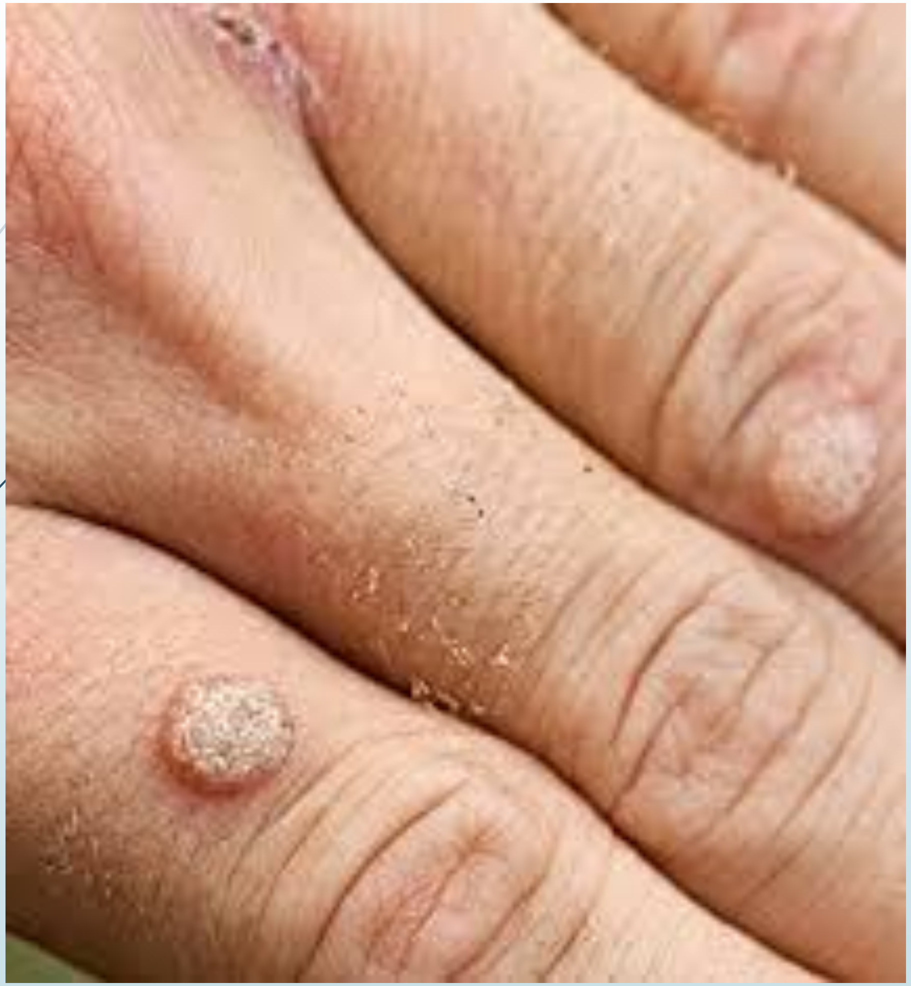
- It’s a common ,discrete benign epidermal hyperplasia induced by different type of human papilloma virus (HPV)
- Wart are very common infection all over the word.
- Both sexes are equally affected.
- All ages but rate in infancy & common wart in school years.
- The highest incidence of common wart is between the age of (9_16)yrs
- While for genital wart between 20_40yrs
- Wart more common in immunocompromised patients.

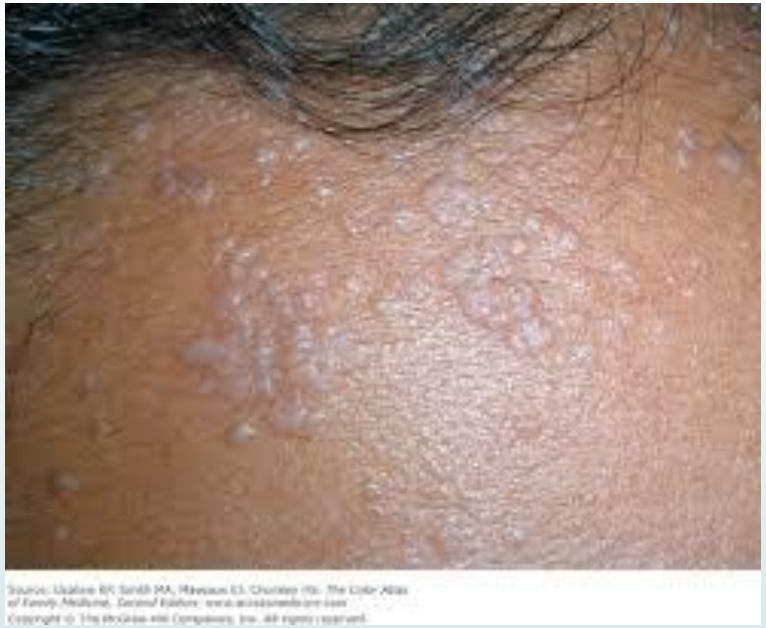
Mode of transmission

- Skin to skin contact (major route)
- minor trauma .
- Habitual nail biter
- Children who suck their hand at risk of auto inculation.
- Shaving may spread wart over the beard
- Genital wart is the commonest STD
- **Cause** : wart caused by HVP
- IP: 1_6 months

CP:

- common wart (verruca vulgaris).
- Plane (flat) wart (verruca plane).
- Planter wart (verruca planters)
- Genital wart (condyloma accuminata)
- Filiform wart
- Periungual wart
- Mosaic wart .
- **Dx**
- History & examination
- PCR
- Pap smear (cervical & anal wart).





Source: Usatine RP, Smith MA, Mayeaux EJ, Chumley HS: The Color Atlas of Family Medicine, Second Edition: www.accessmedicine.com

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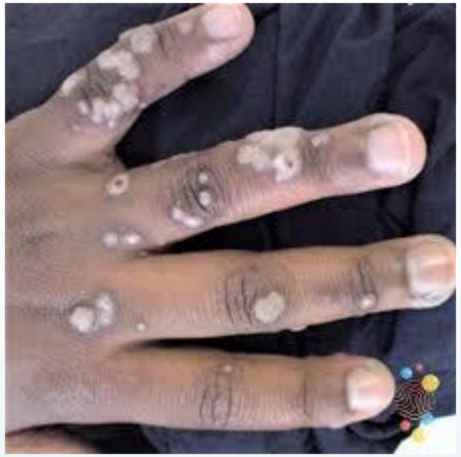
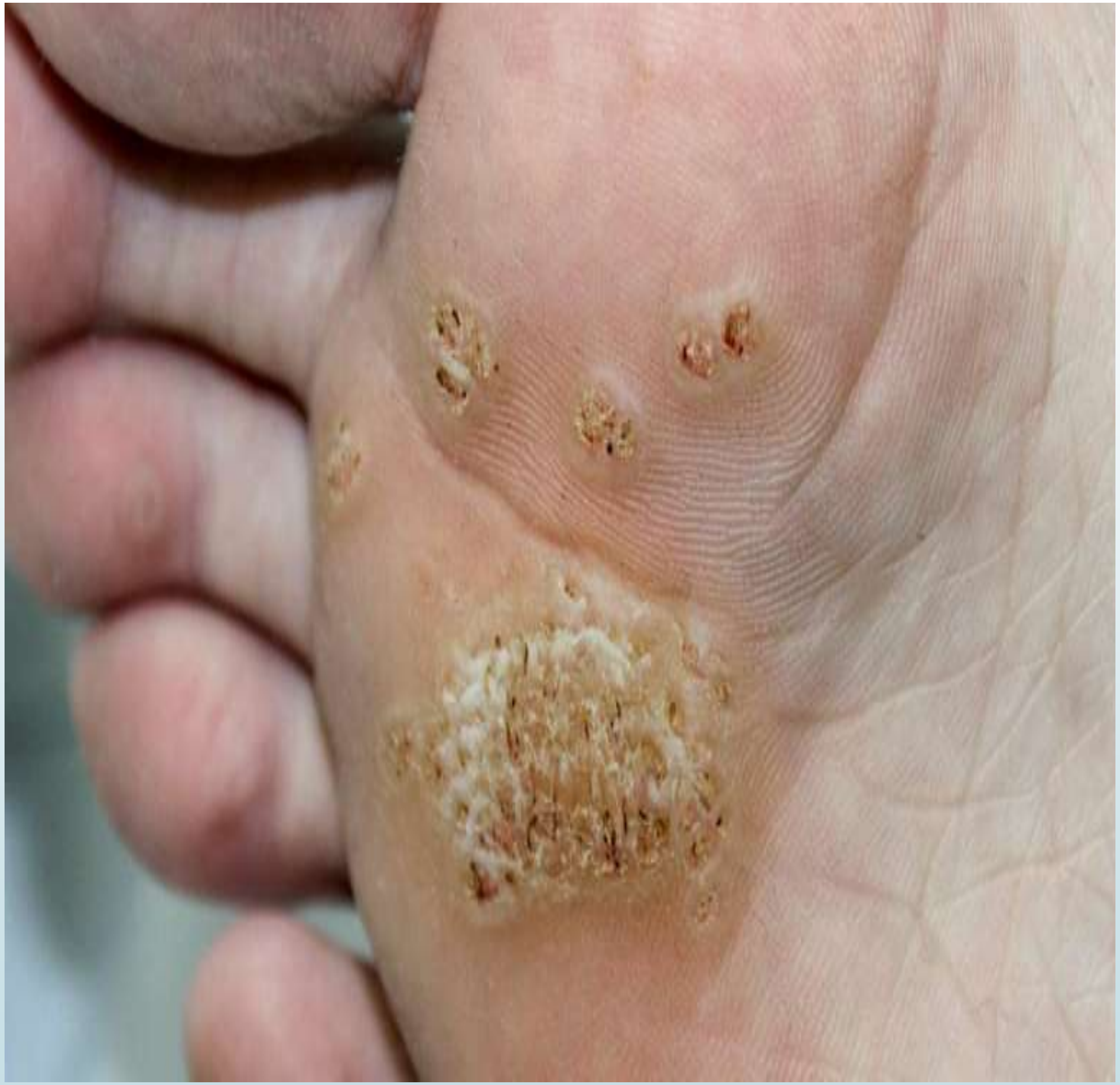
Treatment

- Depend on No of lesions,sites of wart
- And cosmotic disability.
- Keratolytic
- Chemical cautry
- Retinoic acid
- Cryotherapy
- Electrocautry
- Topical 5 fluorouracil
- Podophyllin 20 %
- Imiquimod 5 %
- Laser therapy





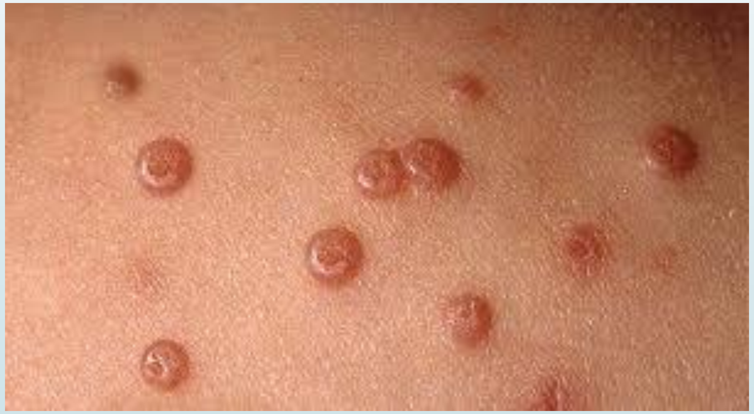
Mollusum contagiosum

- MC is a self limited Viral skin infection
- ch.ch by skin _colored papules that are often umbilicated, occurring in children & sexually active adults
- Both sexes are equally affected.
- In children occur on exposed skin
- In adult may occur in genital skin .
- IP: 4_8 wks

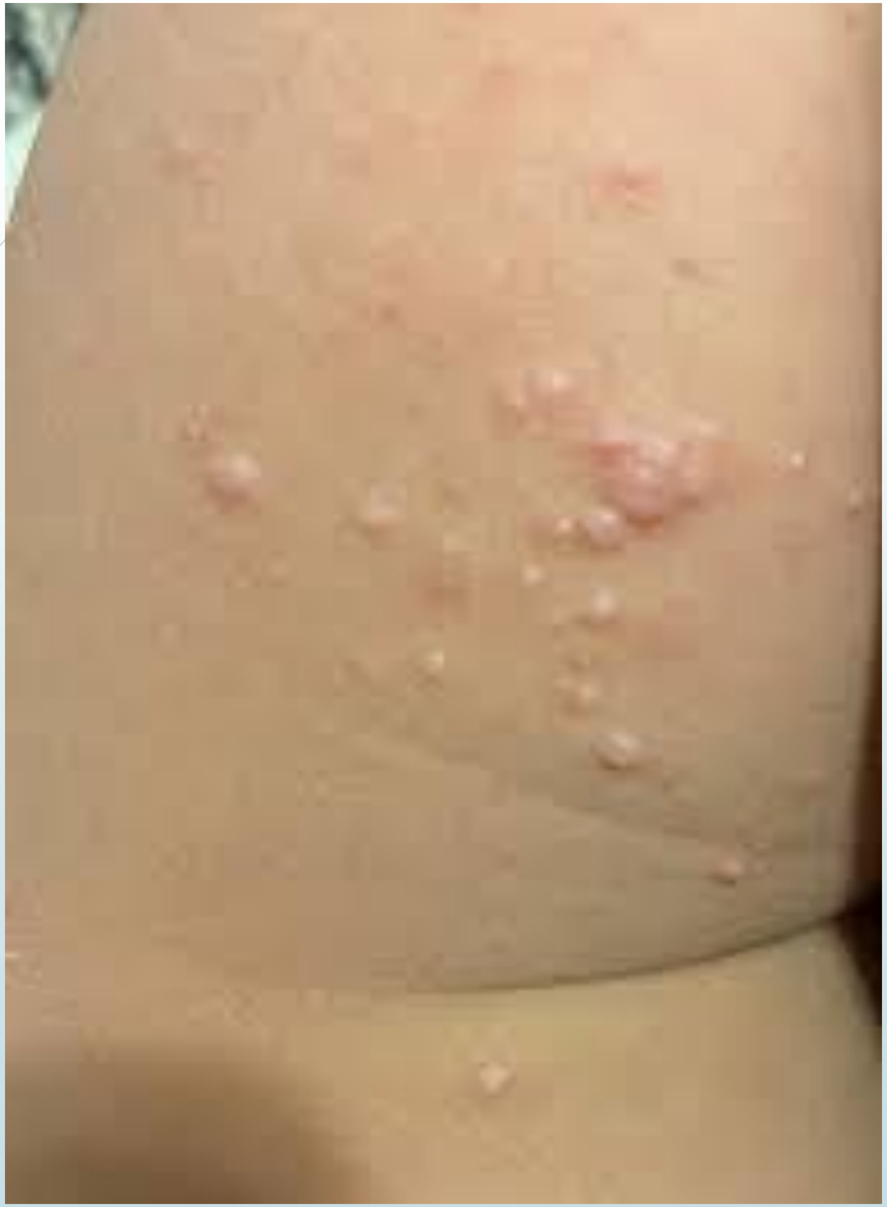
Clinical picutre

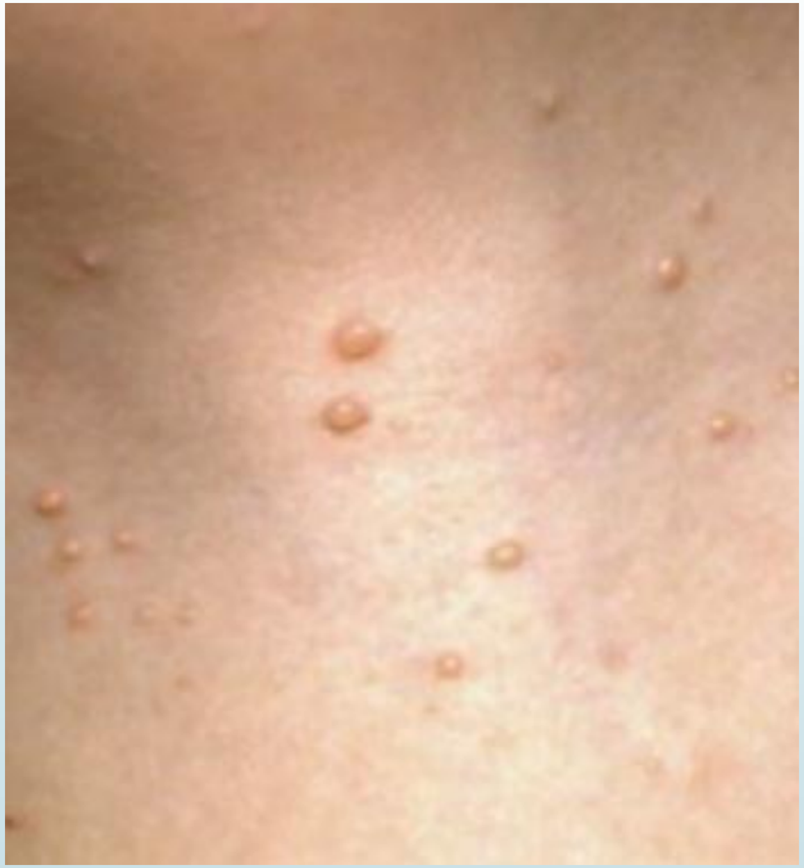
- Lesions is asymptomatic ,pearly while translucent or skin colored papules
- Smooth surface with umblicated center
- Squeezing of lesion will extruded milky white material

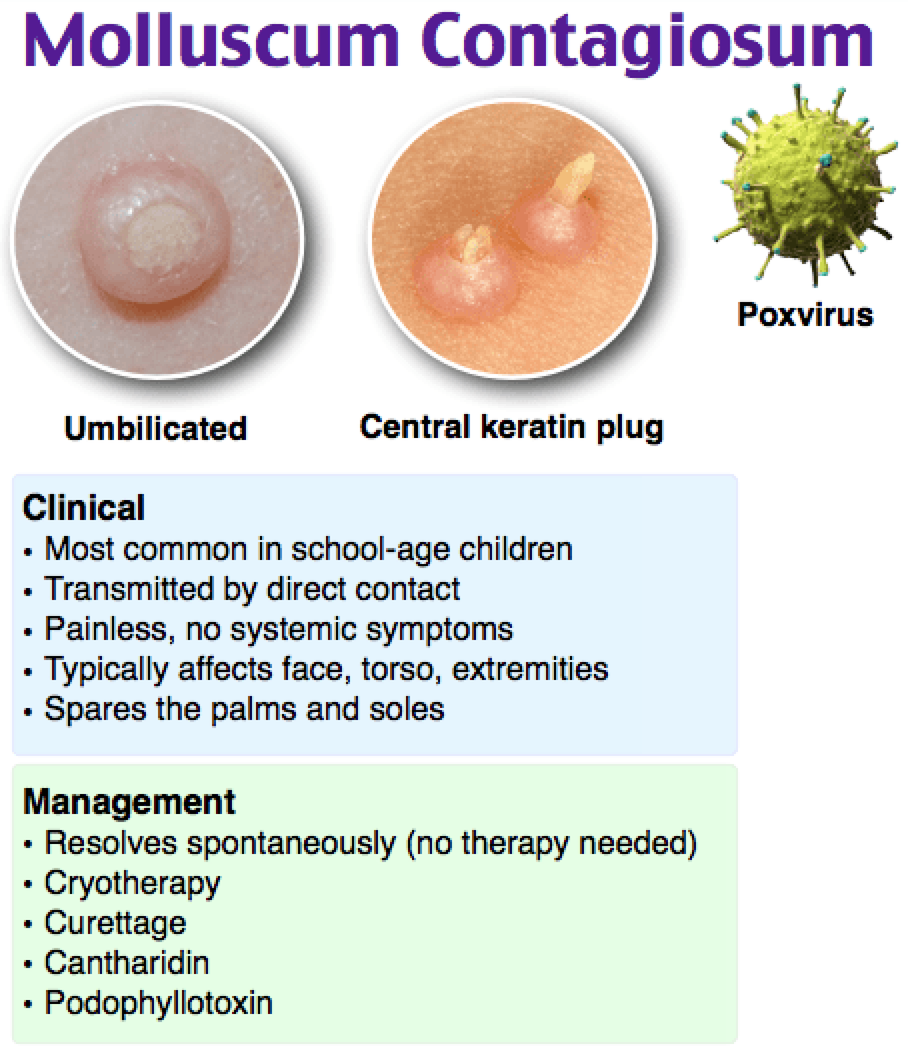
Treatment



- Prevention
- Avoid skin to skin contact with infected individuals
- Curettage.
- Cryotherapy
- Elctrodessiciation
- Topical imiquimod (aldara).







Molluscum Contagiosum

Umbilicated
Central keratin plug
Poxvirus

Clinical
- Most common in school-age children
- Transmitted by direct contact
- Painless, no systemic symptoms
- Typically affects face, torso, extremities
- Spares the palms and soles

Management
- Resolves spontaneously (no therapy needed)
- Cryotherapy
- Curettage
- Cantharidin
- Podophyllotoxin

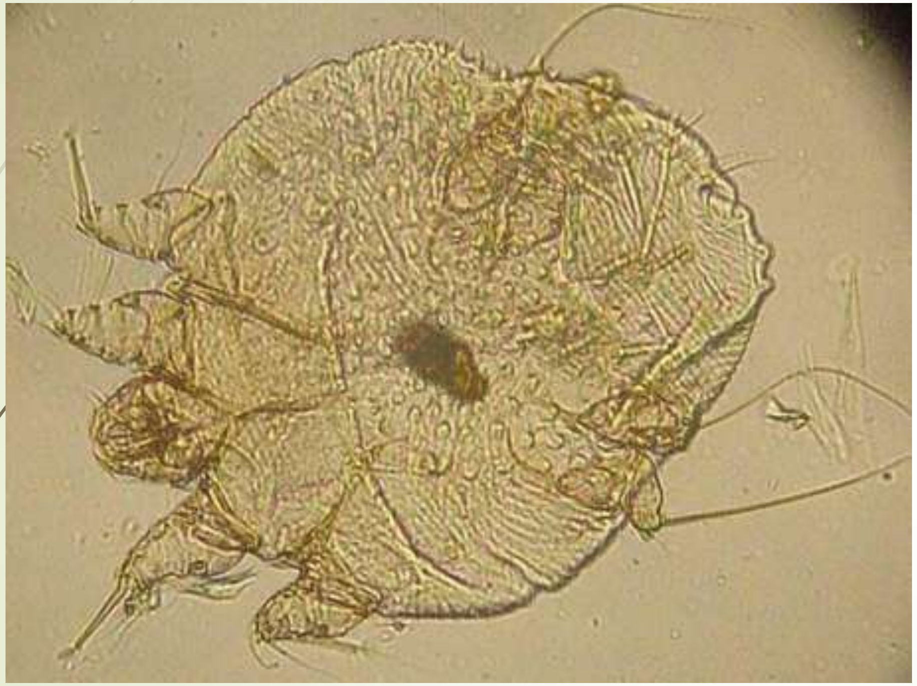
Thank
You

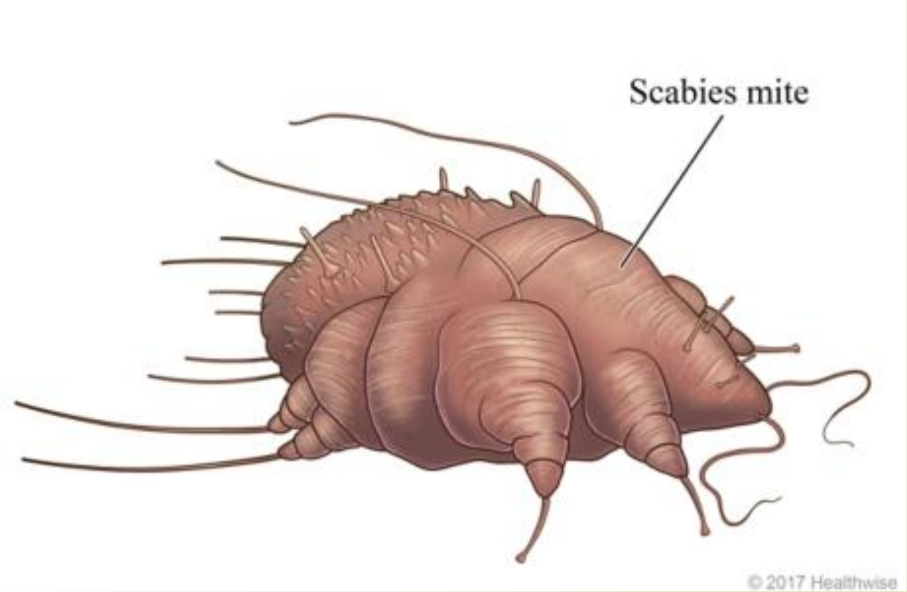
Parasitic infection

Dr :Niveen Almaisary

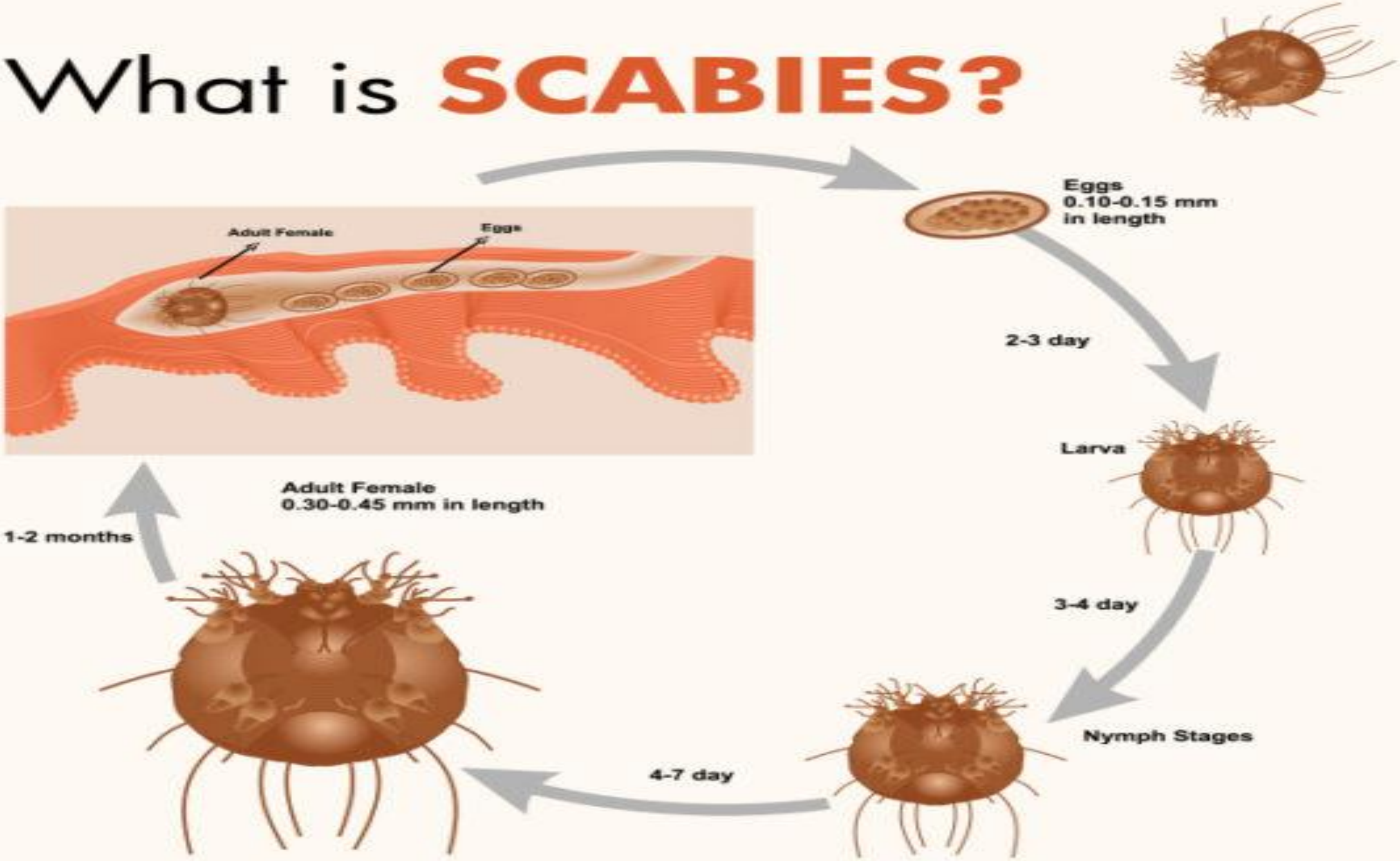
Scabies

- A very common infectious disease caused by a fertilized female mite called sarcoptes scabiei
- It associated with severe itching
- ***Usually transmitted :***
- Direct by close physical contact or indirect : by using clothes or bed of the patient
- Also contact with infected animals
- Away from the host scabies mites survive for 24 – 36hr




Scabies mite
© 2017 Healthwise

What is SCABIES?

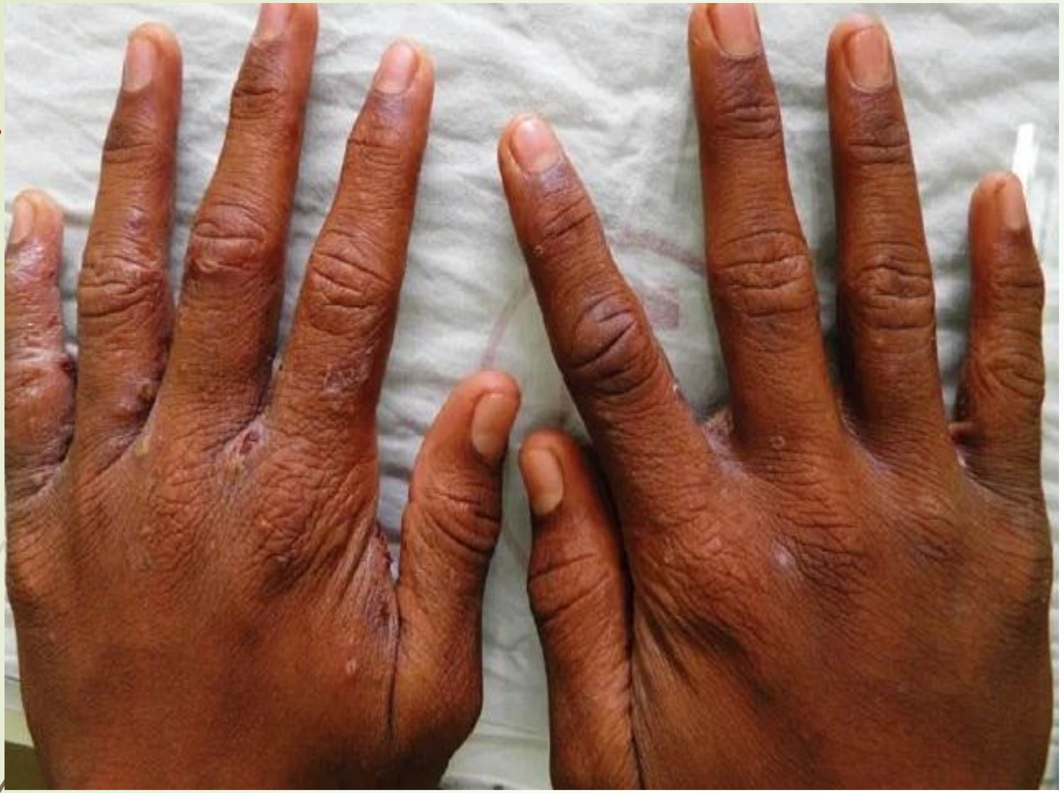

Adult Female
Eggs
Eggs
0.10-0.15 mm
in length
2-3 day
Larva
3-4 day
Nymph Stages
4-7 day
Adult Female
0.30-0.45 mm in length
1-2 months

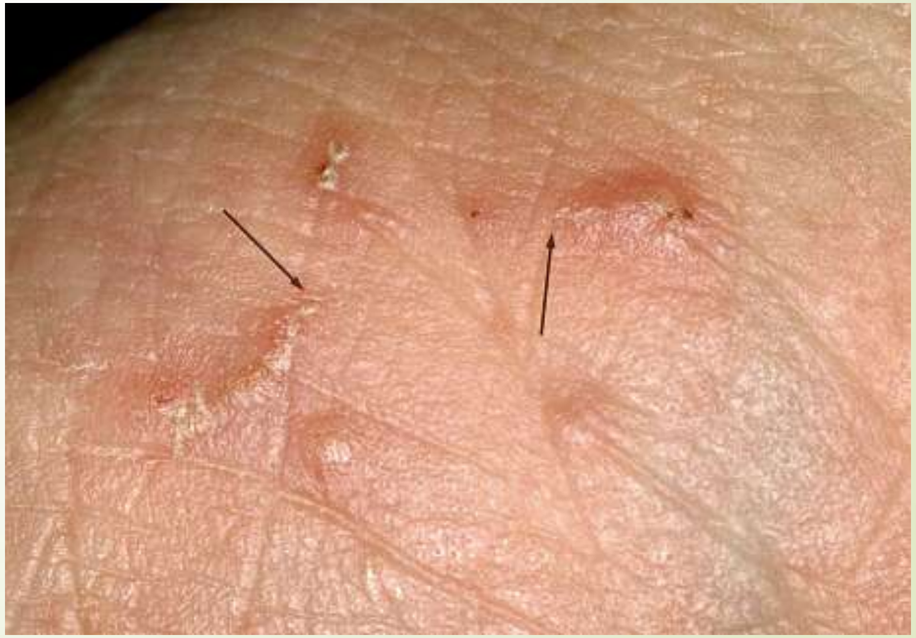
Scabies,cont

- Can occur in any age or and in both sexes
- Itching sever especially at night 3-4 wks after infection occur

Typical locations of lesion :

- Finger webs, the flexor, surface of the wrists, the elbows, the axilla, the buttocks and genitalia and breasts of the women
- The face is often spared
- The typical lesions of scabies are **burrows**





Scabies, cont

- Scabies in infants & young children show more extensive distribution of burrows, vesicularpustular lesions
- Extensive eczematisation is often present
- Multiple crusted nodules on the trunk and limbs

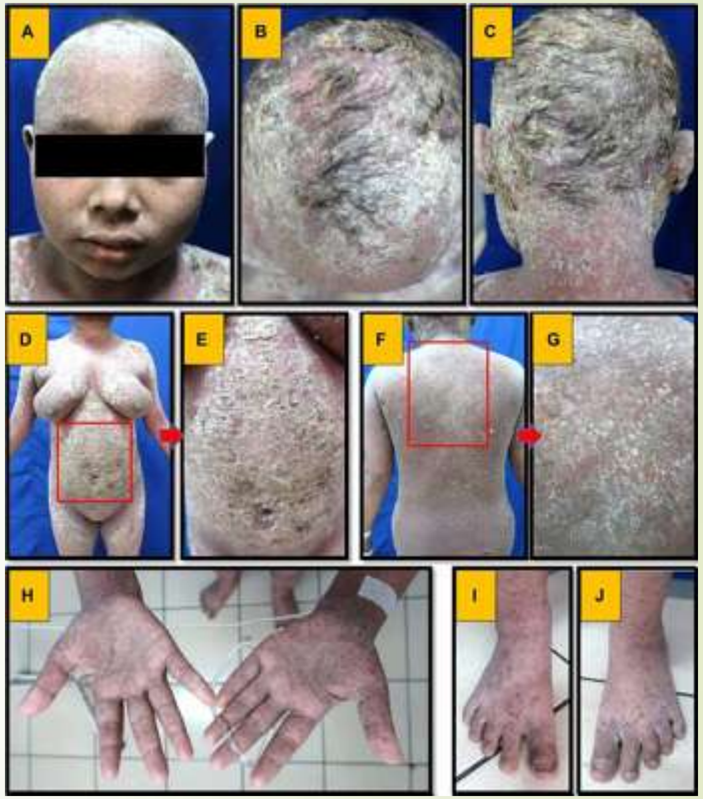
- **Complicalion**
- Secondary bacterial infection
- Eczematous changes
- Acrophobia

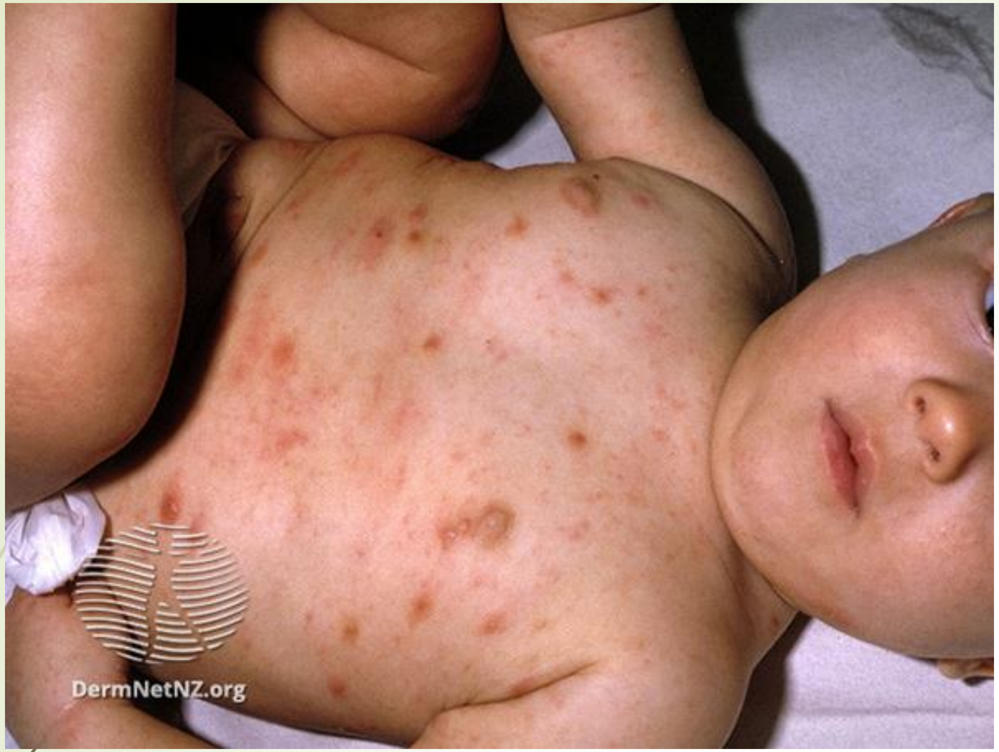
Clinical varient :

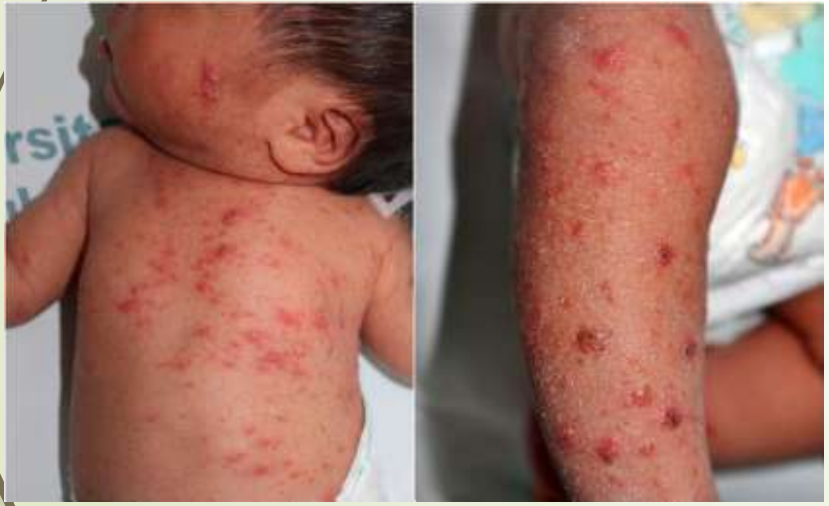
- ❑ Scabies incognito
- ❑ Scabies in clean
- ❑ Nodular scabies
- ❑ Infant scabies
- ❑ Norwegian scabies

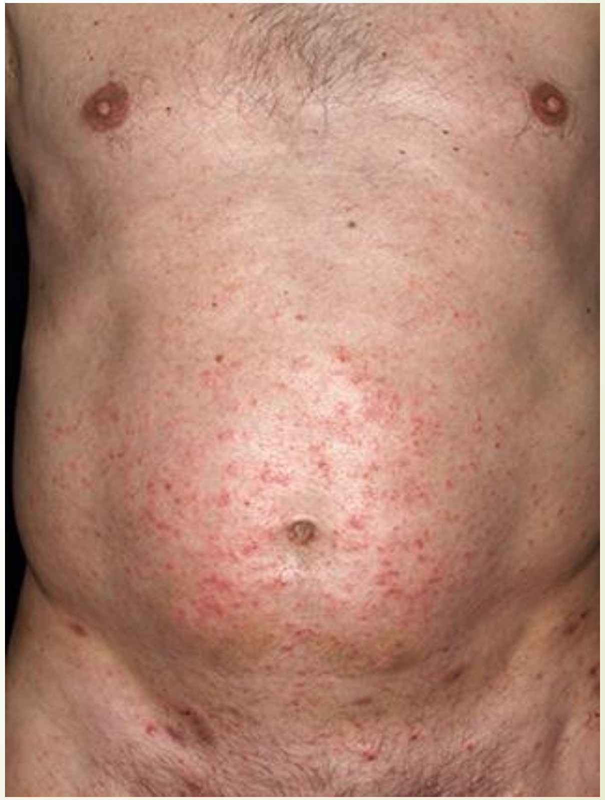
DDx :

- ➢ papular urticaria ,
- ➢ pediculosis corpories
- ➢ Eczema




DermNetNZ.org





Diagnosis :

- Mainly clinical
- Itching mainly at night
- Prsesence of burrows
- Distribution of the lesions
- Contact cases within family
- Demonstration of parasite under microscope

Management :

- **Prophylactic measures :**
- ☐ early diagnosis & treatment
- ☐ Treatment of other family members, disinfection and treatment of infected animals.
- **Medical treatment :**
- ☐ treatment of 2nd infection & 2nd eczema
- ☐ Systemic anti histamine for itching.
- ☐ Scabicidals (topical) :
  - benzyl benzoate 25%
  - Crotamition 10%
  - Gama benzenc hexacloride 1%

Scabies. Cont,

  - Permethrin cream 2.5% for child&5%for adult
- Oral ivermectin for sever & resistance cases.
- Intralesional Steriod injection for post scabietic nodules

Causes of post scabietic pruritus

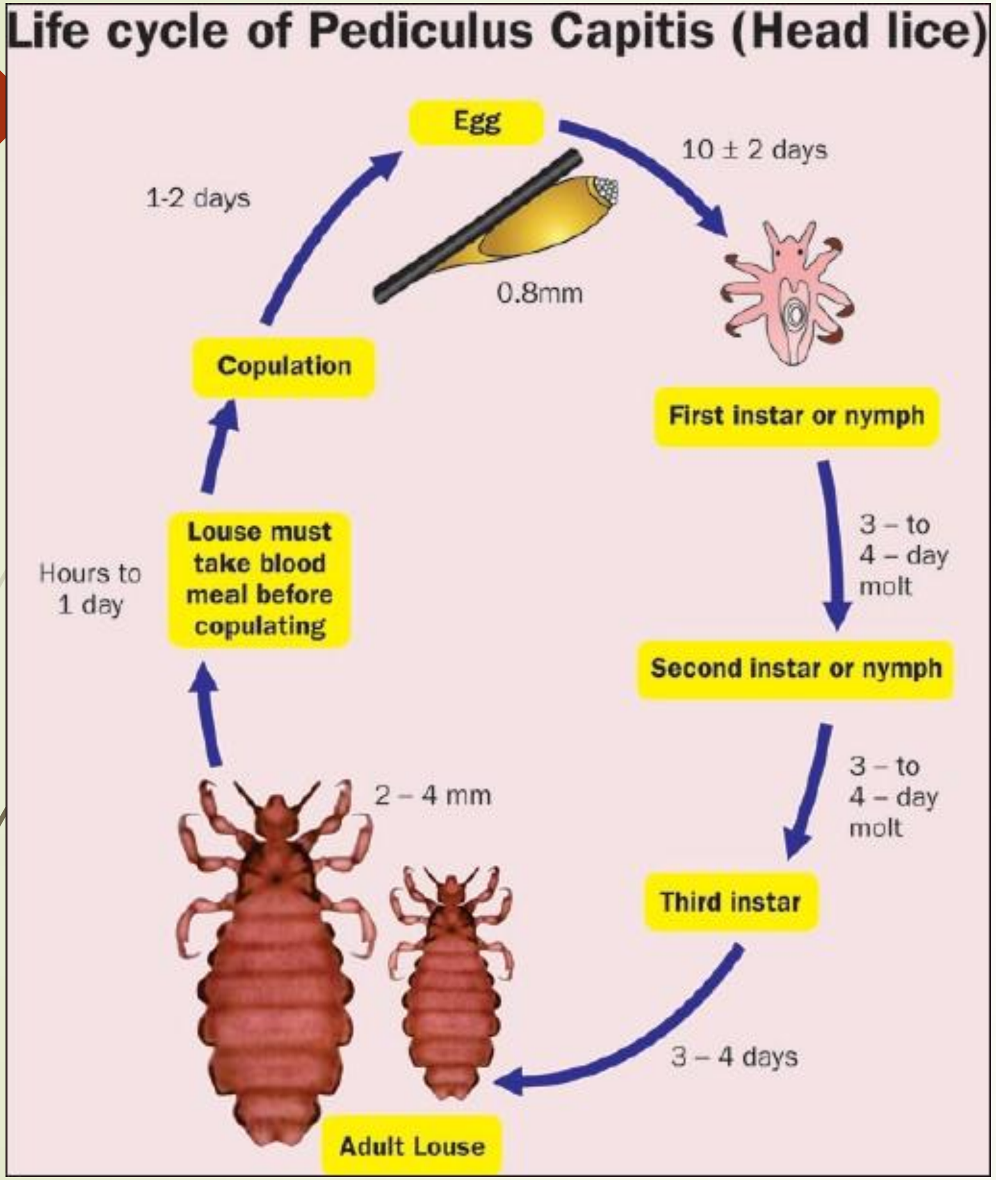
- ❑ Irritation of skin by topical preparation
- ❑ Post scabietic nodules, pruritus
- ❑ acarophobia

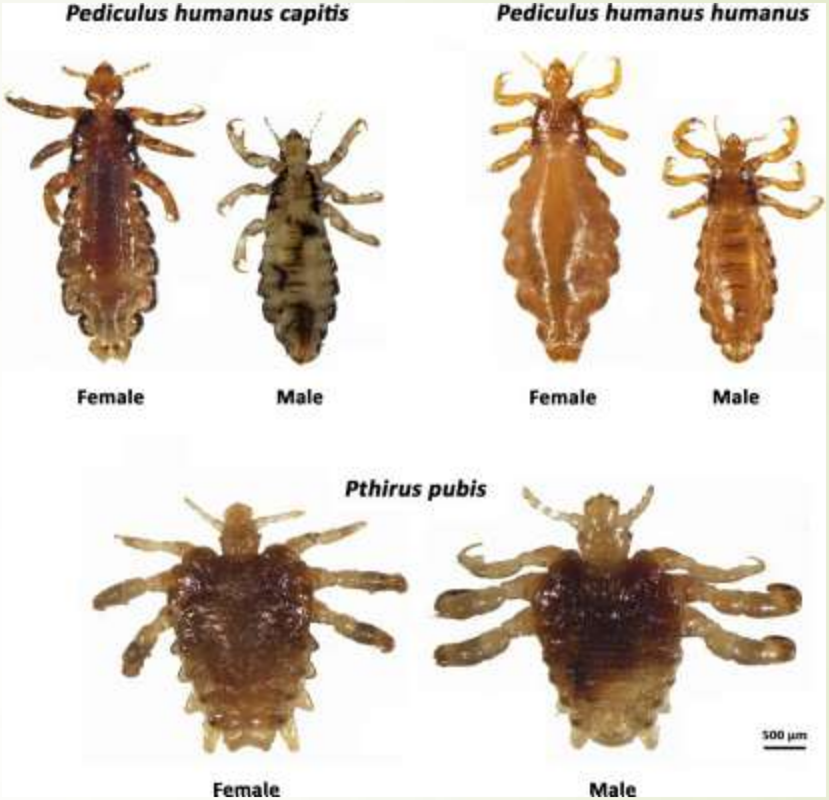
lice

- Lice are insects that live on human hair and clothing.
- Lice are wingless and have 6 legs on which are attached strong claws, which they use to grasp on tightly to hair shafts or clothing fibres.
- Head lice are the most common infestation in humans
- their eggs are called nits
- The medical term for an infection or infestation with lice is called "**pediculosis**

There are 3 types of lice that infest humans.

- 1. Pediculus humanus var. capitis: The head louse.
- 2. Pediculus humanus var. humanus
- Phthirus pubis: The pubic louse.

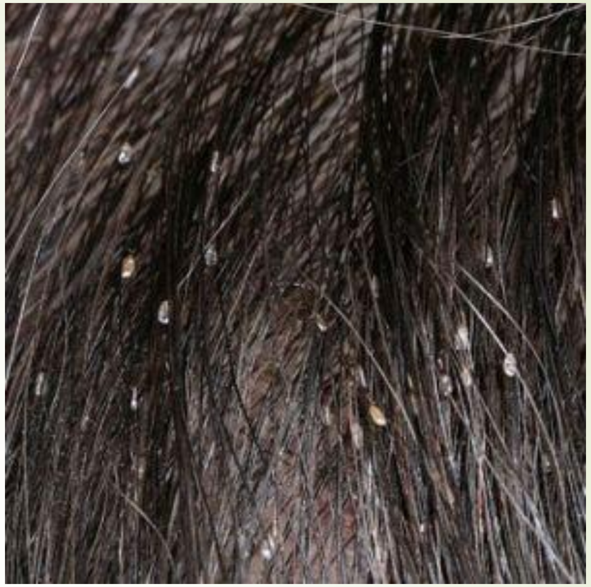

Life cycle of Pediculus Capitis (Head lice)
Egg
10 ± 2 days
1-2 days
0.8mm
Copulation
First instar or nymph
3 – to 4 – day molt
Hours to 1 day
Louse must take blood meal before copulating
Second instar or nymph
3 – to 4 – day molt
2 – 4 mm
Third instar
3 – 4 days
Adult Louse


Pediculus humanus capitis
Pediculus humanus humanus
Female
Male
Female
Male
Pthirus pubis
500 µm
Female
Male

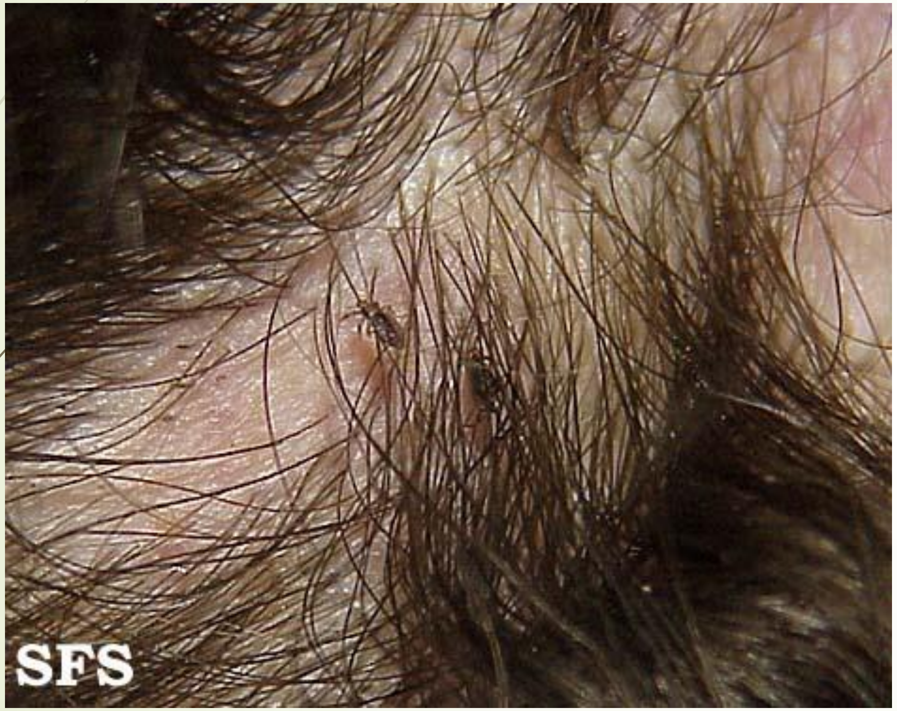
Pediculosis capitis

- The usual organism is **Pediculus humanus capitis**,
- very prevalent especially in school children and crowded areas in most societies.
- Occurs most commonly in children 3–11 years, girls > boys
- The majority of infestations are acquired by direct head to head contact
- The louse reaches maturity in approximately 10 days.
- Head lice feed on human blood and **itching** from lice bites is a common symptom of this condition
- (normally intensifies 3-4 weeks after the initial infestation).
- Scalp pruritus is characteristic,

Pediculosis capitis



- some may be asymptomatic
- Secondary bacterial infection may occur from scratching
- Pruritic papular lesions may occur on the nape of the neck, lymphadenopathy
- concomitant head louse infection must always be considered in cases of scalp impetigo
- The empty egg cases occur in greatest density on the parietal and occipital regions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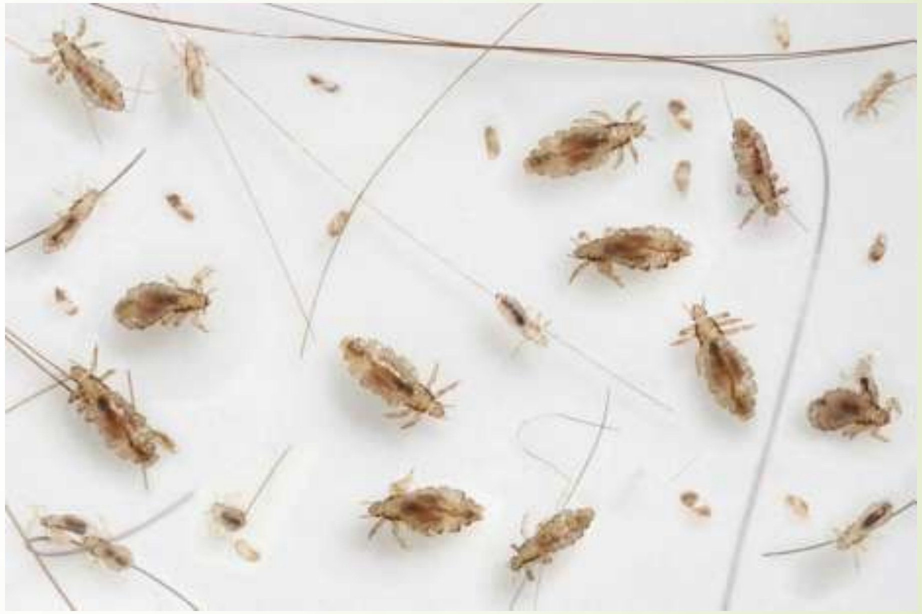


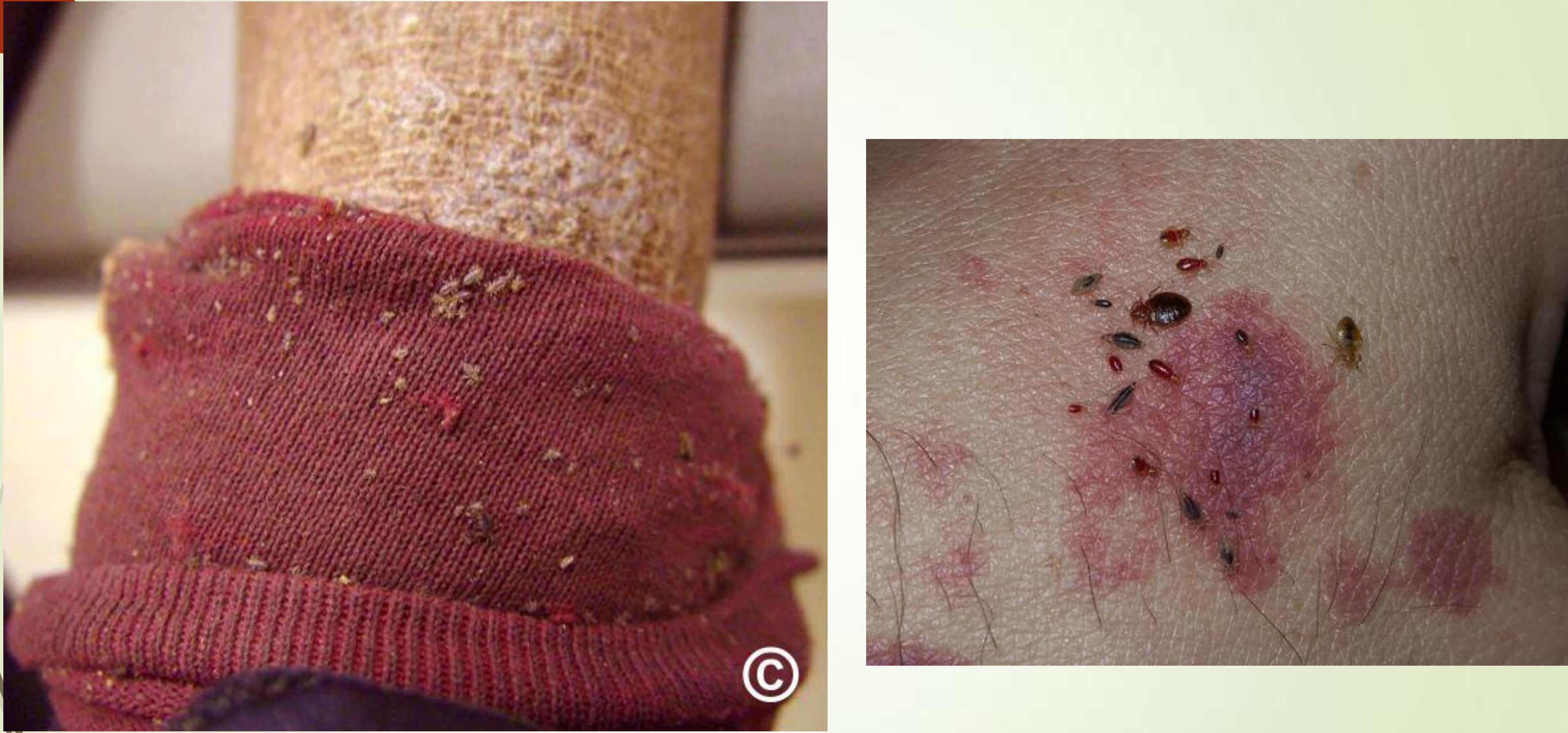
Management

- Live head lice can be treated by wet combing with fine-toothed comb;
- chemical treatment if ineffective, e.g malathion,permtherin
- Treat all affected contacts and repeat after 7–10 days

Causes of therapeutic failure for head lice include ***misunderstanding of instructions, non-adherence, resistance, failure to retreat after 7–10 days and reinfestation***

Clothing/body lice (Syn. Pediculus corporis)

- associated with poor socioeconomic conditions.
- The natural habitat is the clothing of its host, and lice only visit the skin to feed. Eggs are cemented to clothing fibers
- **Clinical features**
- Itching is the main complaint.
- The body is often covered in excoriations
- and/or secondary bacterial infection.
- Post-inflammatory pigmentation occurs in longstanding disease
- Lice and eggs should be sought in the clothing



Management

- Bed linens and clothes should be decontaminated by hot washing.
- Infested materials sealed in plastic bags may be used safely after 3 days.
- Infested furniture, mattresses and box springs should be discarded or fumigated to destroy lice and nits

Crab lice (Syn. Phthiriasis pubis

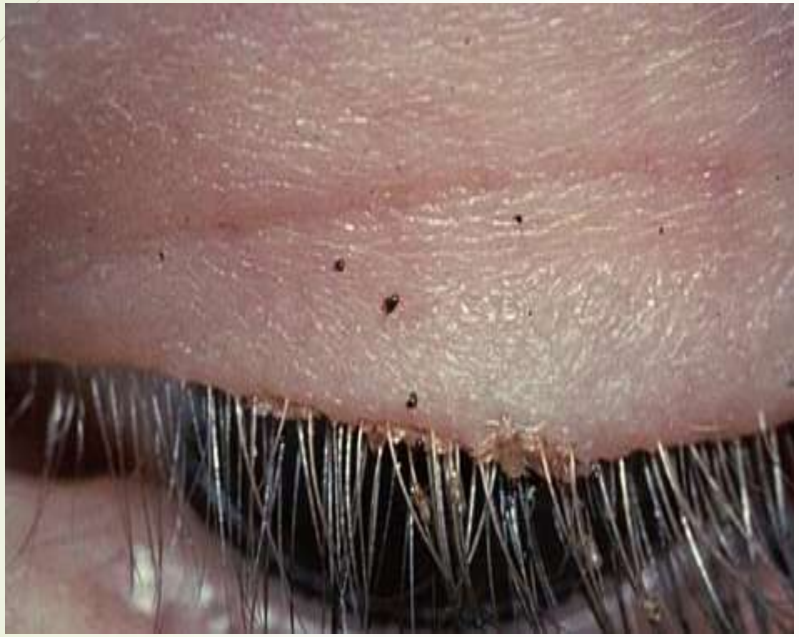
- known as Phtirus pubis.
- contracted by close contact and are easily transmitted sexually.
- The pubic hair is most common site
- but lice can spread to other hairy parts of the body including axilla, beard, chest hair and thigh hair.
- Eyelashes can also be affected

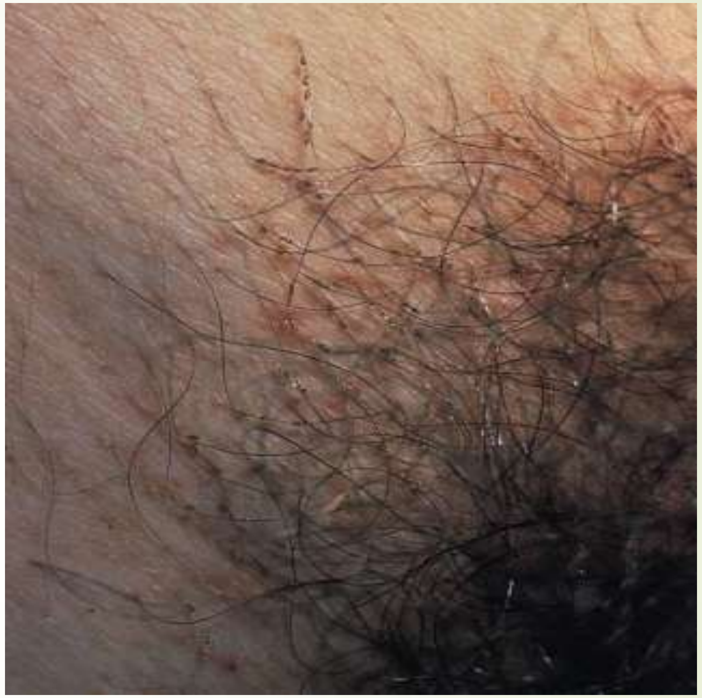
Clinical features

- Itching, mainly in the evening and at night,

is the main symptom.

- Inspection of affected areas will reveal lice on hairs close to the skin surface,
- and louse eggs attached to the hair shafts
- When pubic crab lice are identified other hairy areas of the body should be examined

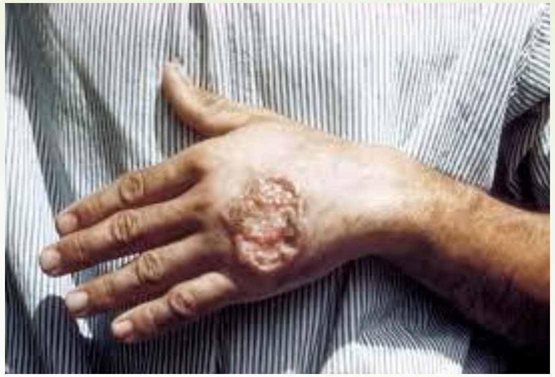




Management

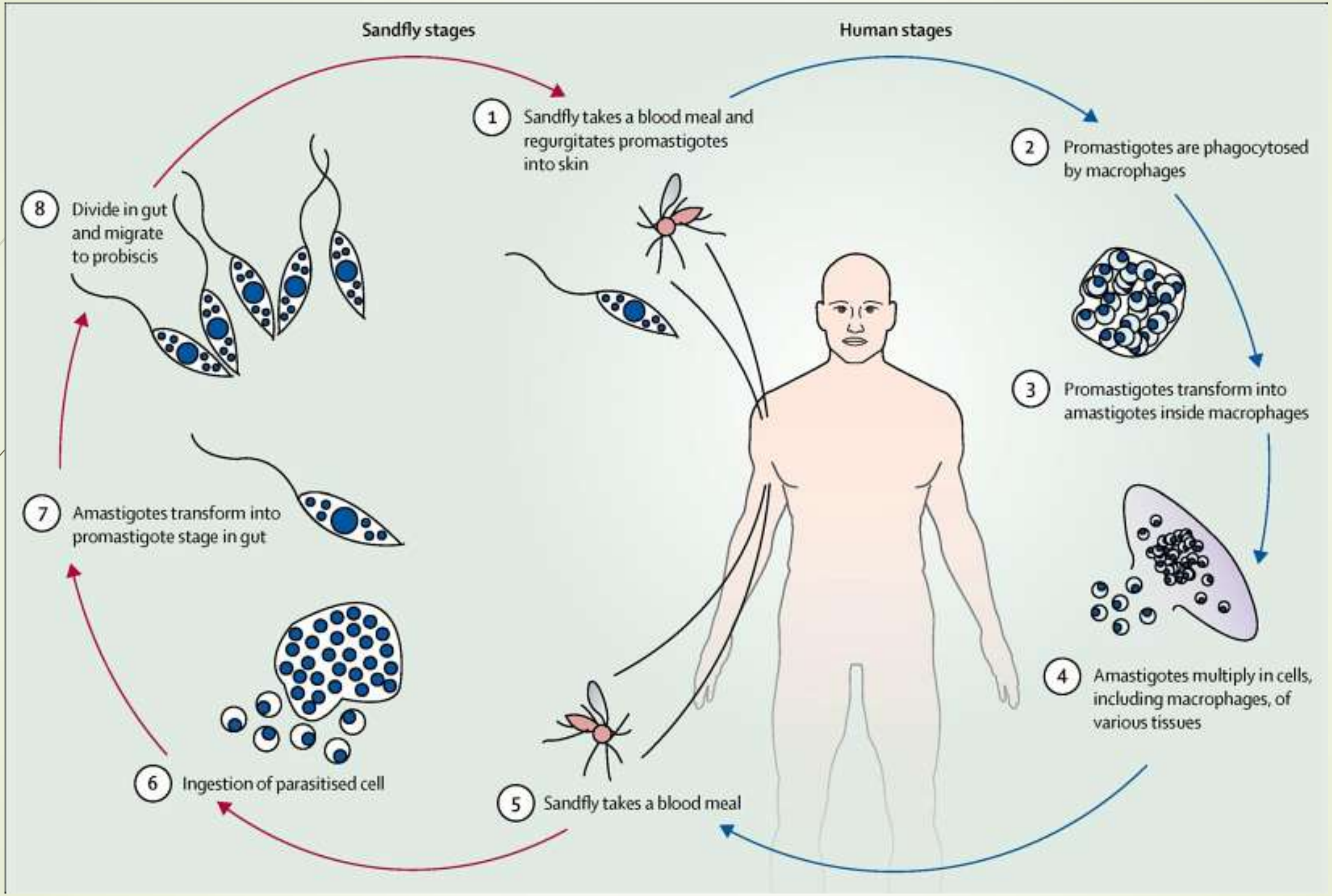
- All hairy areas of the body should be treated at the same time.
- Pubic lice are treated with the same insecticidal creams or lotions as pediculosis capitis.
- with a second application after 7–10 days.
- Eyelash infestations should be treated with permethrin 5% cream (washed off after 10 min) or only with petrolatum (applied twice a day for 8–10 days),
- followed by mechanical removal of the nits.
- Screen for associated sexually transmitted disease

Leishmaniasis



- is a disease caused by protozoan parasites that belong to the genus Leishmania
- and is transmitted by the bite of certain species of sand fly.
- Most forms of the disease are transmissible only from animals (zoonosis), but some can be spread between humans.
- Human infection is caused by about 21 of 30 species that infect mammals.
- One of the most common forms of the disease is cutaneous leishmaniasis that occurs most commonly ,over 90%

- **Life Cycle**The organism is transmitted by the bite of several species of blood-feeding sand flies (Phlebotomus)
- which carry the **promastigote** in the anterior gut and pharynx.
- The parasites gain access to mononuclear phagocytes where they transform into **amastigotes** and divide until the infected cell ruptures.
- The released organisms infect other cells.
- The sandfly acquires the organisms during the blood meal; the amastigotes transform into flagellate promastigotes and multiply in the gut until the anterior gut and pharynx are packed. Dogs and rodents are common reservoirs

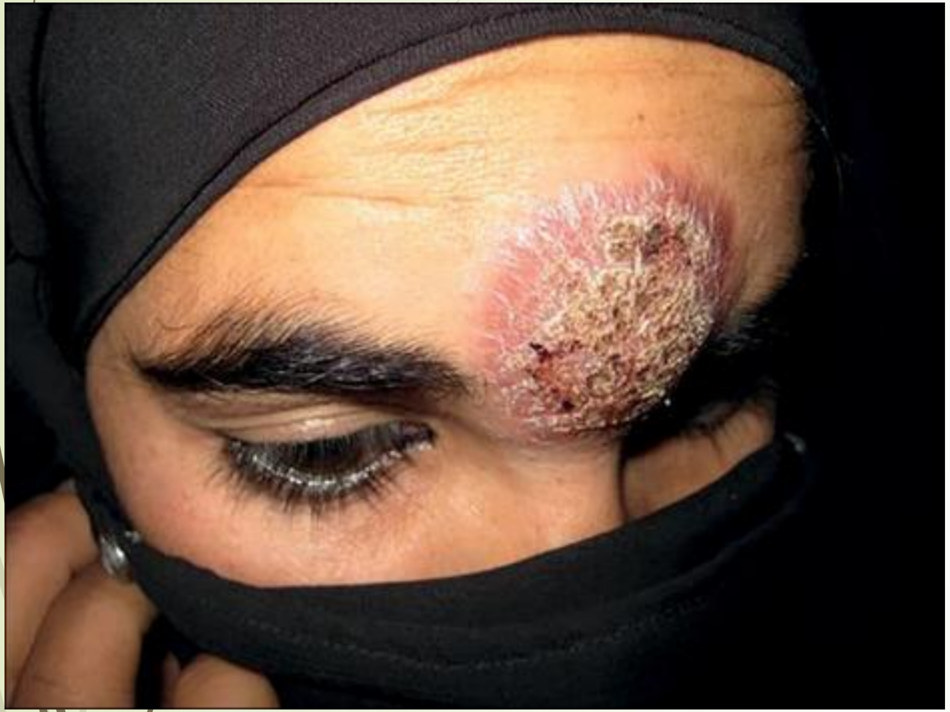


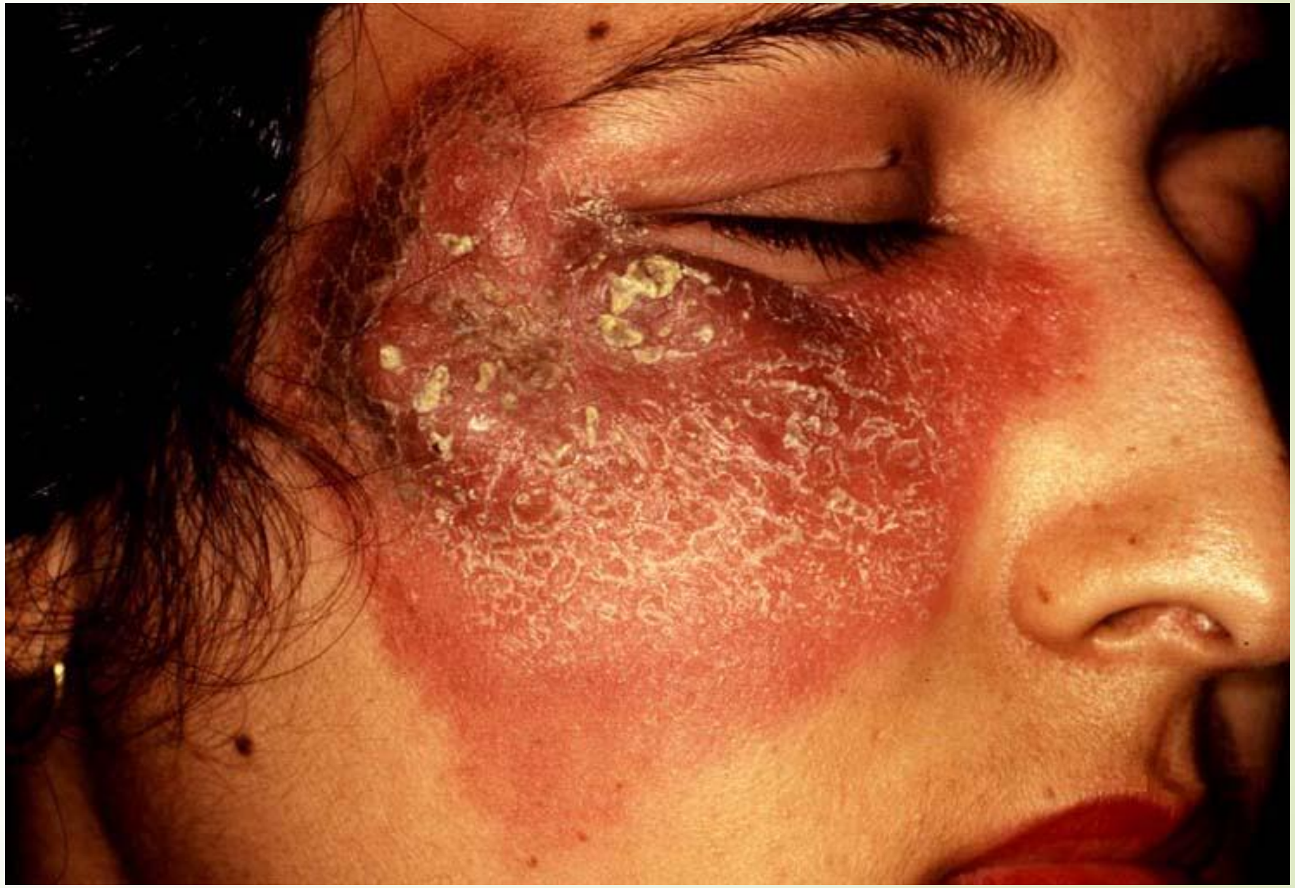
1. Sandfly takes a blood meal and regurgitates promastigotes into skin
2. Promastigotes are phagocytosed by macrophages
3. Promastigotes transform into amastigotes inside macrophages
4. Amastigotes multiply in cells, including macrophages, of various tissues
5. Sandfly takes a blood meal
6. Ingestion of parasitised cell
7. Amastigotes transform into promastigote stage in gut
8. Divide in gut and migrate to probiscis

- .. **Visceral leishmaniasis** is a severe form in which the parasites have migrated to the vital organs.
- The most important species of Leishmania is L. donovani causes visceral leishmaniasis (Kala-azar, black disease, dumdum fever)
- . Leishmaniasis is divided into 4 main clinical forms and is caused by parasitic protozoa of the genus Leishmania

Cutaneous leishmaniasis:

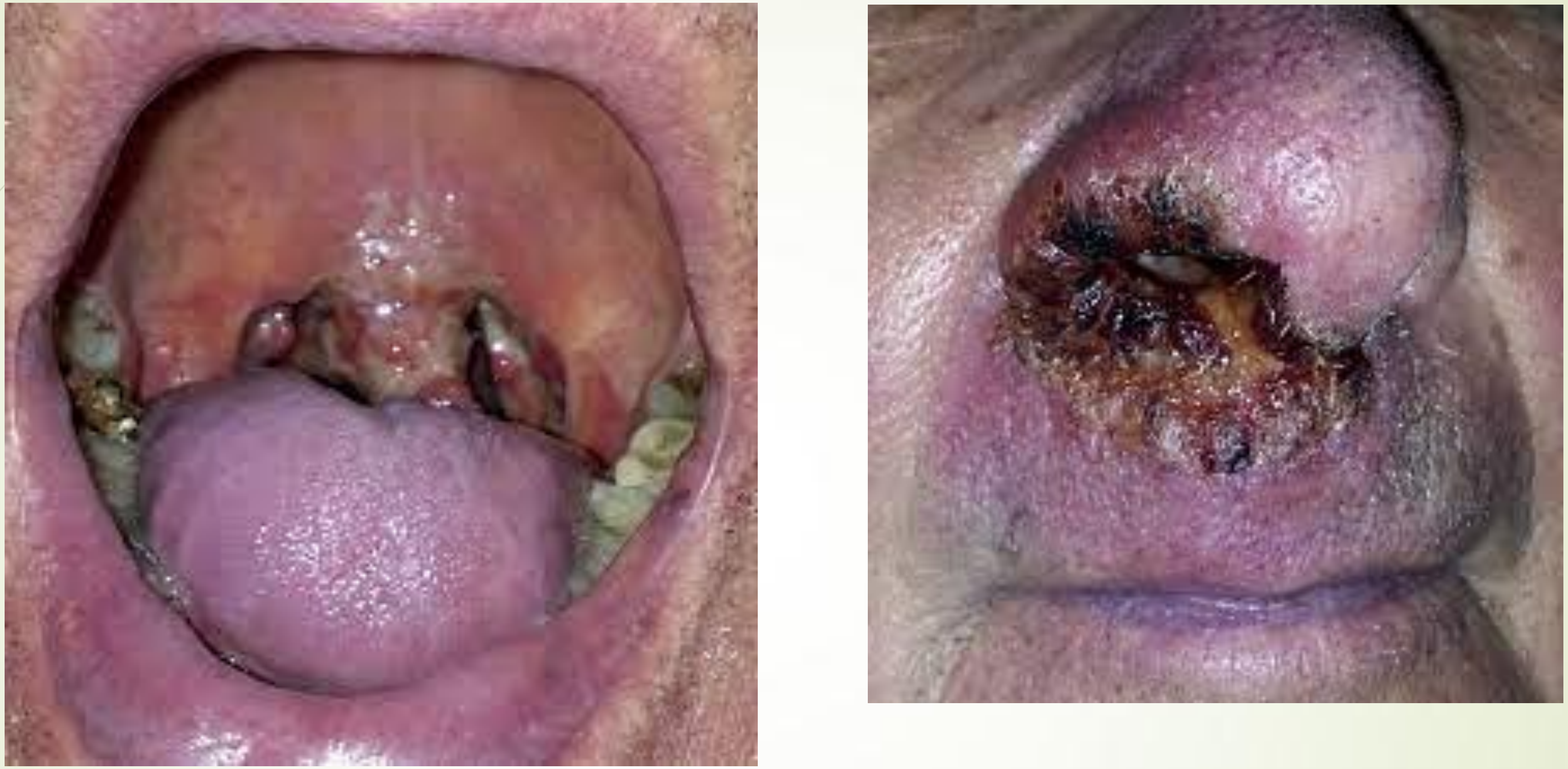
- Cutaneous leishmaniasis is the most common form causing one or more ulcers on the skin.
- start a small red papule up to 2 cm in diameter
- Over several weeks, papules become darker and form ulcers with raised edges and central crater.
- Ulcers can be moist and exude pus or dry with a crusted scab.
- on exposed areas of the skin, especially the face and extremities.
- Lesions may occur immediately after the bite or after weeks or months before causing any ulcers





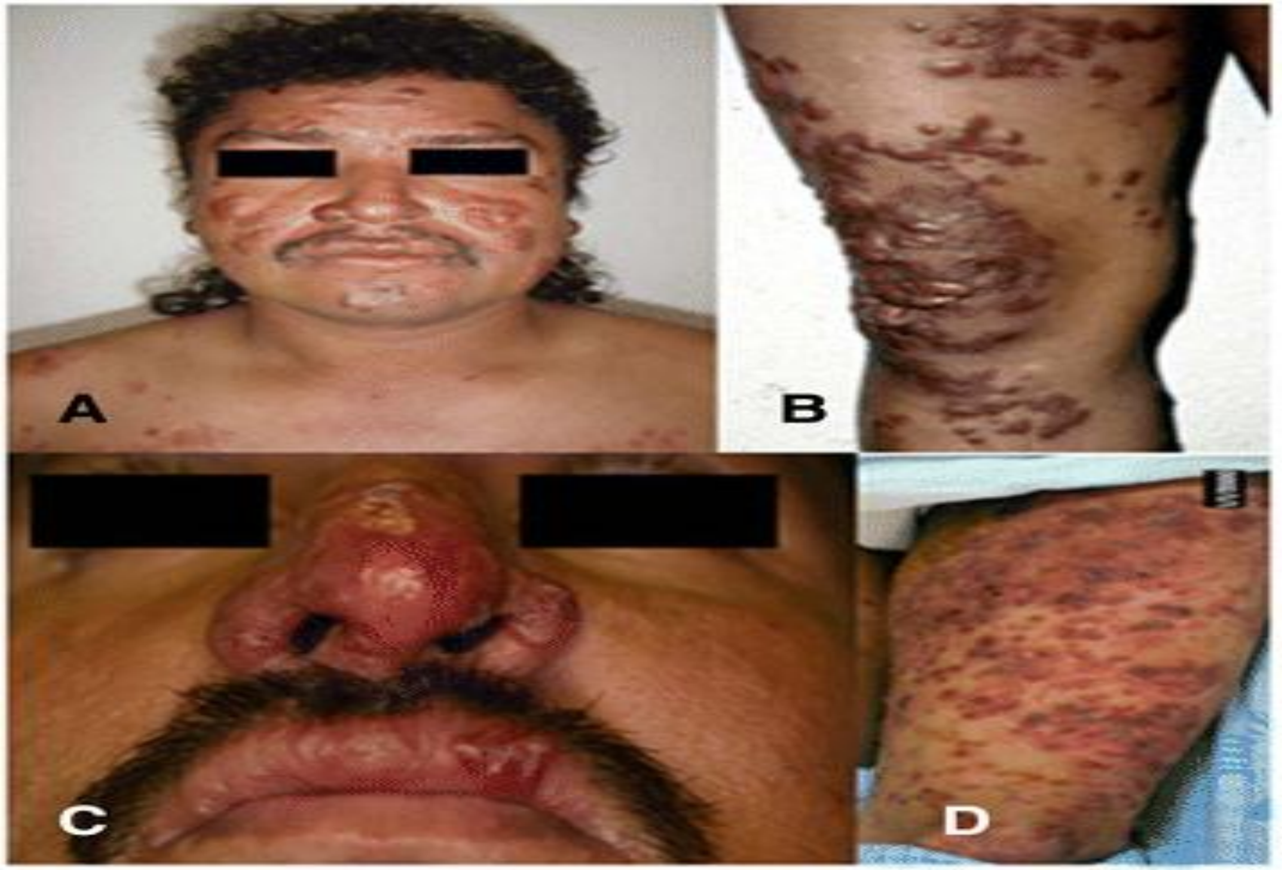
Mucocutaneous leishmaniasis:

- Lesions appearing 1-3 m months after being bitten.
- Ulcers m ay only occur around the mucous membranes of the nose and mouth or may also involve skin lesions
- without treatment, the entire nasal mucosa and palates become deformed with ulceration and erosion of the nasal septum, lips and palate
- Mucosal lesions often are painful and become e sites of infection, sometimes leading to sepsis.



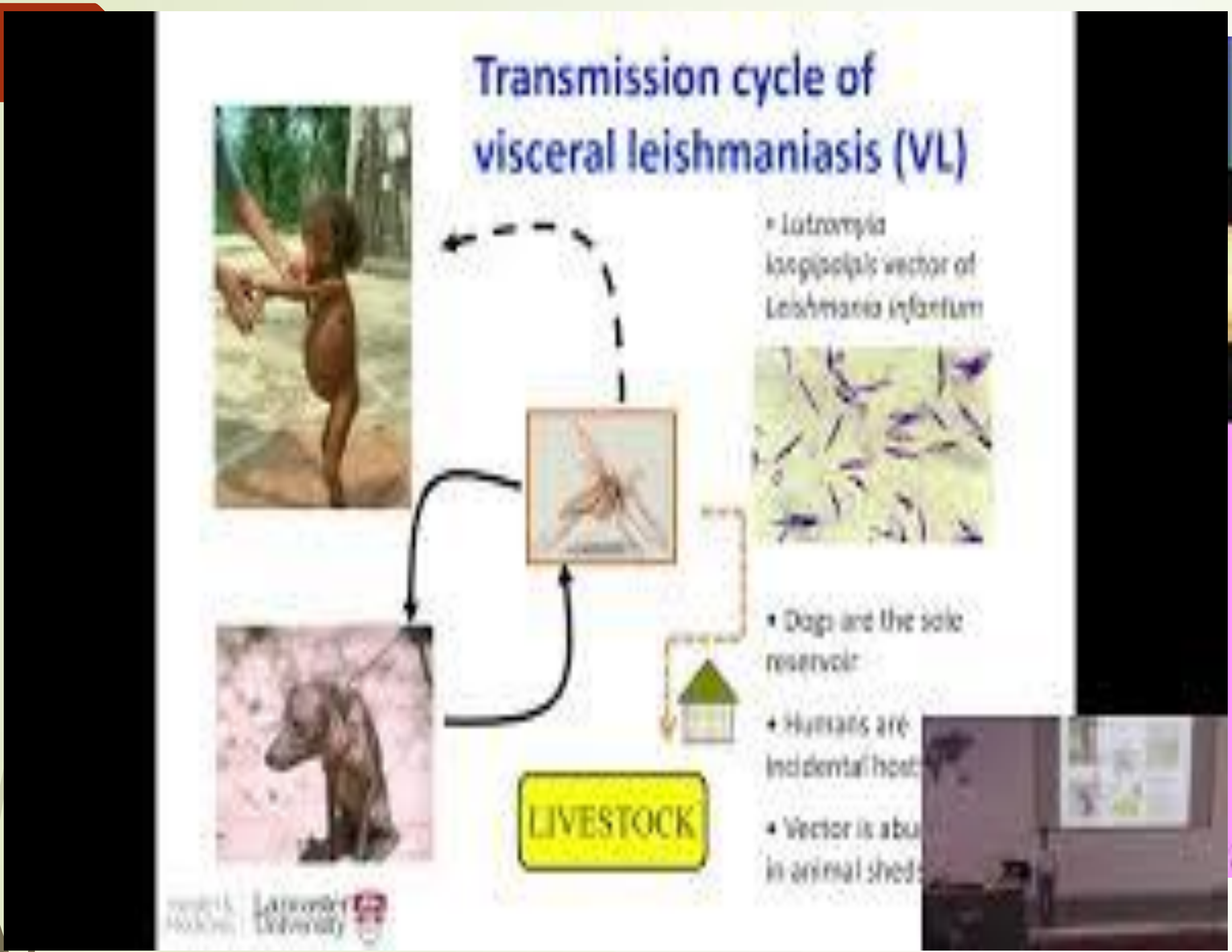
Diffuse cutaneous leishmaniasis

- Disseminated and chronic skin lesions resembling lepromatous leprosy.
- Initial skin lesion spreads locally ,and from which the disease disseminate to other parts of the skin, often involving large area .
- Lesion are nodules that don't ulcerate .
- The disease progresses slowly and become chronic and difficult to treat.


A
B
C
D

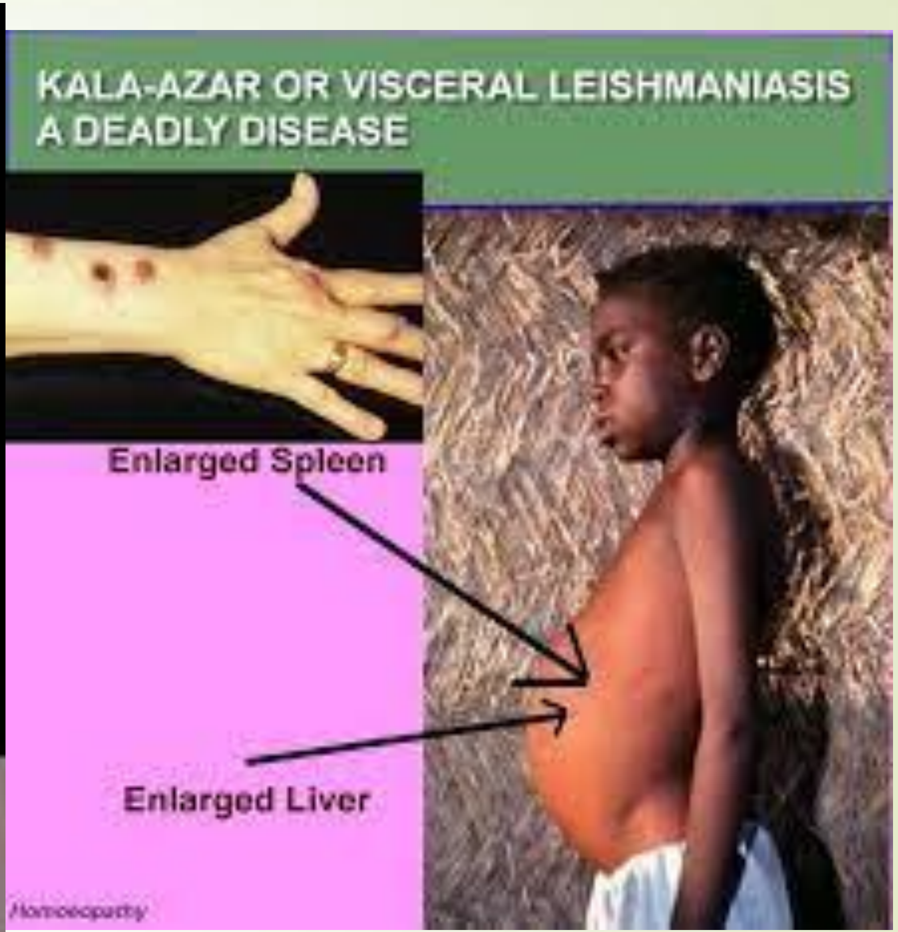
Visceral leishmaniasis:

- affects internal organs including the spleen, liver and lymph nodes.
- It is also known as “**Kala azar**’; because of the characteristic darkening (pigmentation) of the skin in patients with the disease.
- Symptoms include bouts of fever, weakness, diarrhea, swollen glands.
- Hepatosplenomegaly is common.
- The disease may take different forms ranging from asymptomatic self-resolving disease to fulminant, life-threatening disease



Transmission cycle of visceral leishmaniasis (VL)

- Lutzomyia longipalpis vector of Leishmania infantum
- Dogs are the sole reservoir
- Humans are incidental host
- Vector is abu
in animal sheds



KALA-AZAR OR VISCERAL LEISHMANIASIS A DEADLY DISEASE

Enlarged Spleen
Enlarged Liver
Homoeopathy

Diagnosis:

- Leishmaniasis is diagnosed in the haematology laboratory by direct visualization of the **amastigotes** (Leishman-Donovan bodies).
- Buffy-coat preparations of peripheral blood or aspirates from marrow, spleen, lymph nodes or skin lesions should be spread on a slide to make a thin smear, and stained with Leishman's or Giemsa's stain (pH 7.2) for 20 minutes.
- Amastigotes are seen with monocytes or, less commonly in neutrophil in peripheral blood and in macrophages in aspirates

Treatment :

- Most cases of **cutaneous leishmaniasis** resolve spontaneously without treatment, but this can take months or even years. Ulcers often leave unsightly scars
- There are two common therapies containing antimony (known as pentavalent antimonials): meglumine antimoniate (Glucantime) and sodium stibogluconate (Pentostam).
- Liposomal amphotericin B is effective in treating **visceral leishmaniasis**
- Secondary infections should be treated with appropriate antimicrobial therapy.
- Other treatments sometimes used for leishmaniasis include: Azole antifungal drugs: itraconazole, fluconazole, ketoconazole, zinc sulfate, and cryotherapy

THANK
YOU

Nail Disorders

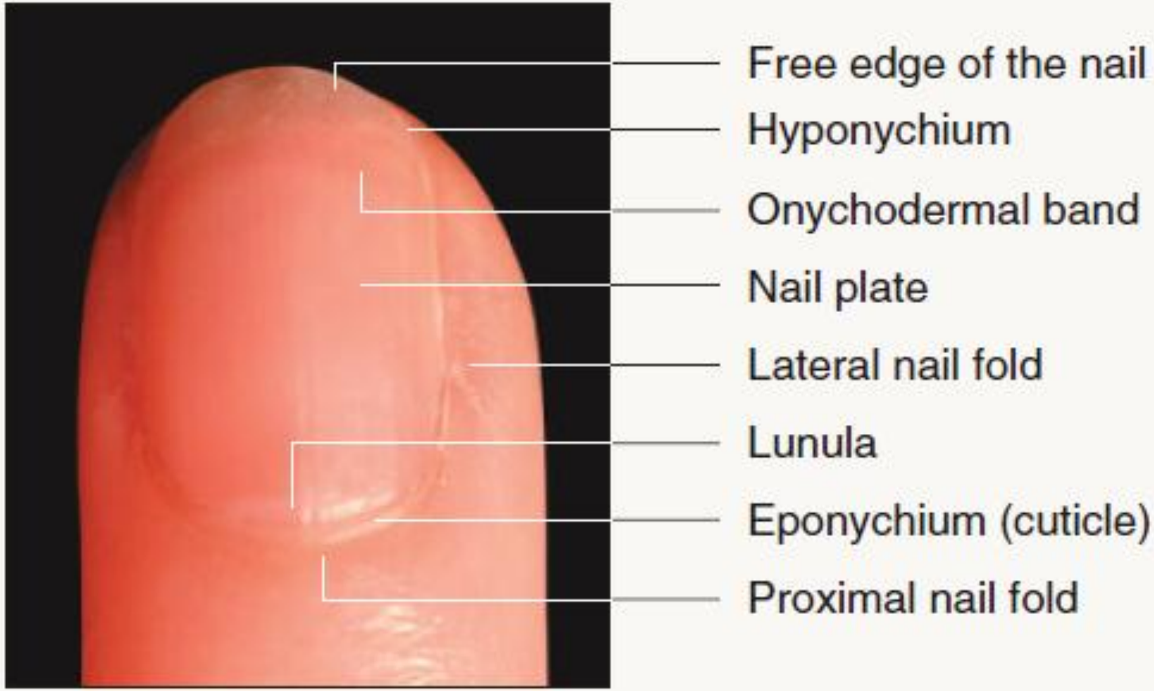
Asst. Prof, Al-Muriesh Maher

Nail disorders

Learning objectives

- Understand the anatomy of the nail organ
- Recognize nail changes
- Congenial & Hereditary nail diseases
- The nail in dermatological diseases
- The nail in systemic diseases

NAIL UNIT ANATOMY


Free edge of the nail
Hyponychium
Onychodermal band
Nail plate
Lateral nail fold
Lunula
Eponychium (cuticle)
Proximal nail fold

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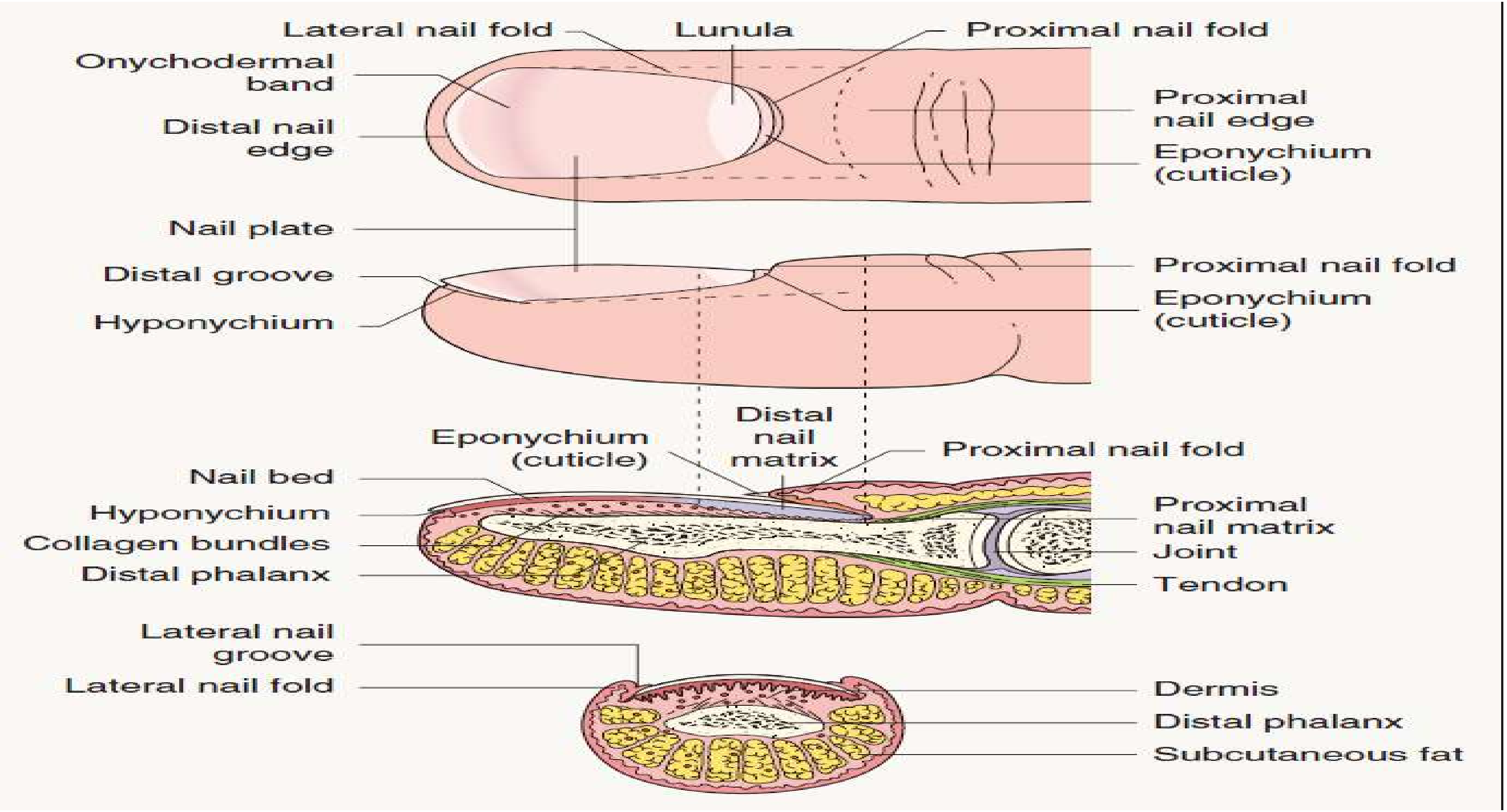
Lateral nail fold
Lunula
Proximal nail fold
Onychodermal band
Distal nail edge
Proximal nail edge
Eponychium (cuticle)
Nail plate
Distal groove
Hyponychium
Proximal nail fold
Eponychium (cuticle)
Eponychium (cuticle)
Distal nail matrix
Proximal nail fold
Nail bed
Hyponychium
Collagen bundles
Distal phalanx
Proximal nail matrix
Joint
Tendon
Lateral nail groove
Lateral nail fold
Dermis
Distal phalanx
Subcutaneous fat

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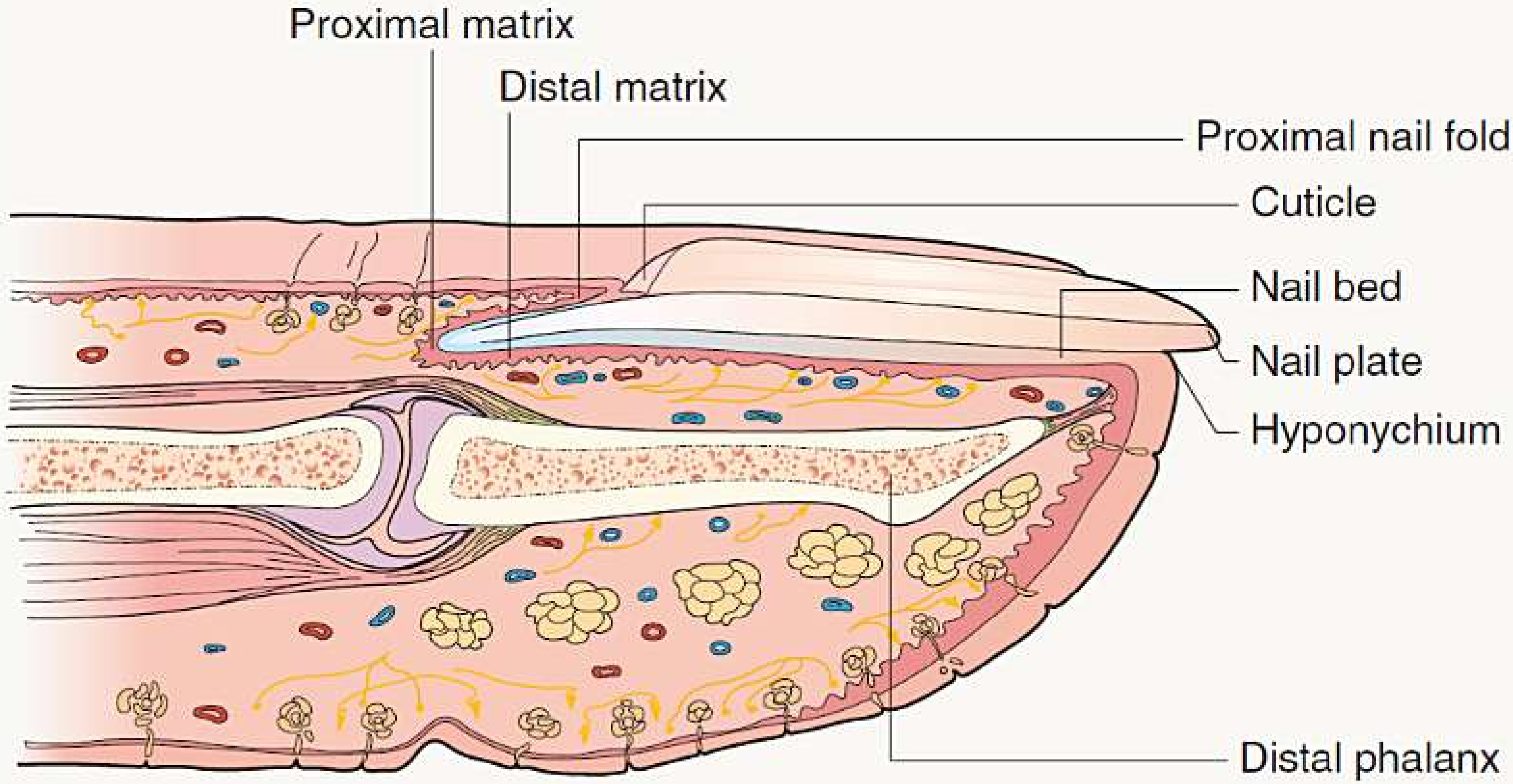
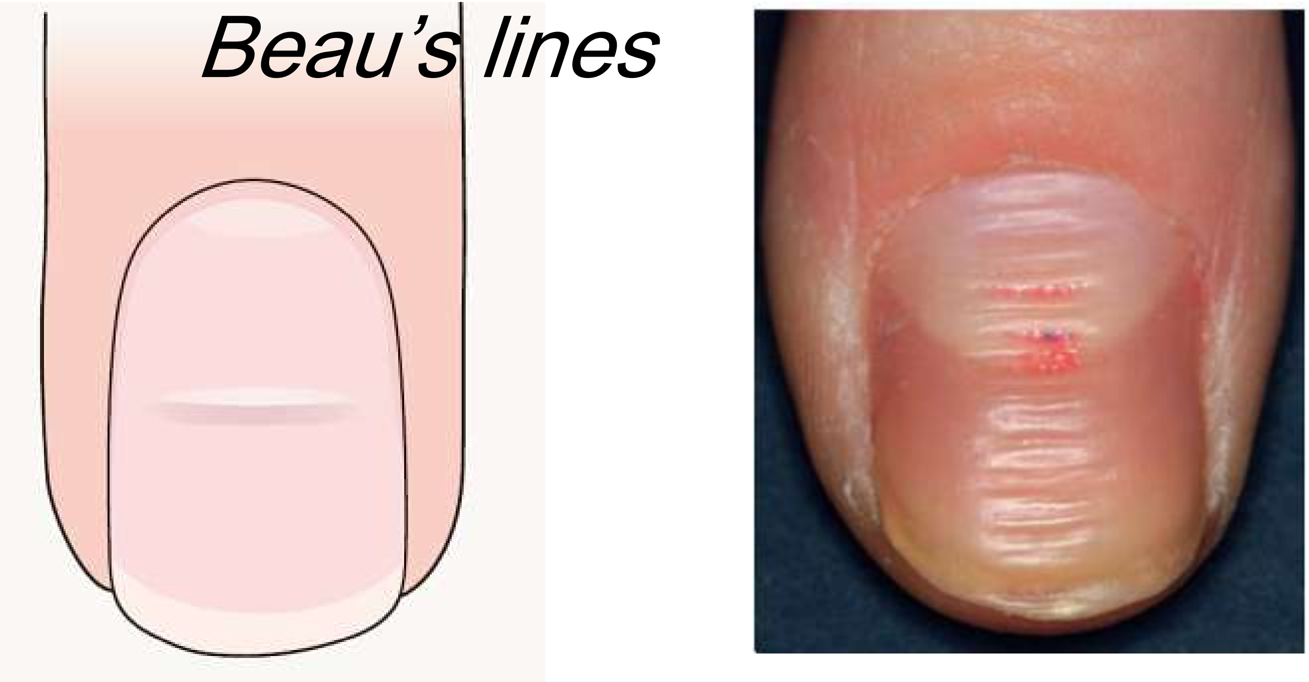
THE NAIL APPARATUS


Proximal matrix
Distal matrix
Proximal nail fold
Cuticle
Nail bed
Nail plate
Hyponychium
Distal phalanx

Nail disorders

Nail Signs Due to Abnormal Nail Matrix Function

Beau’s lines



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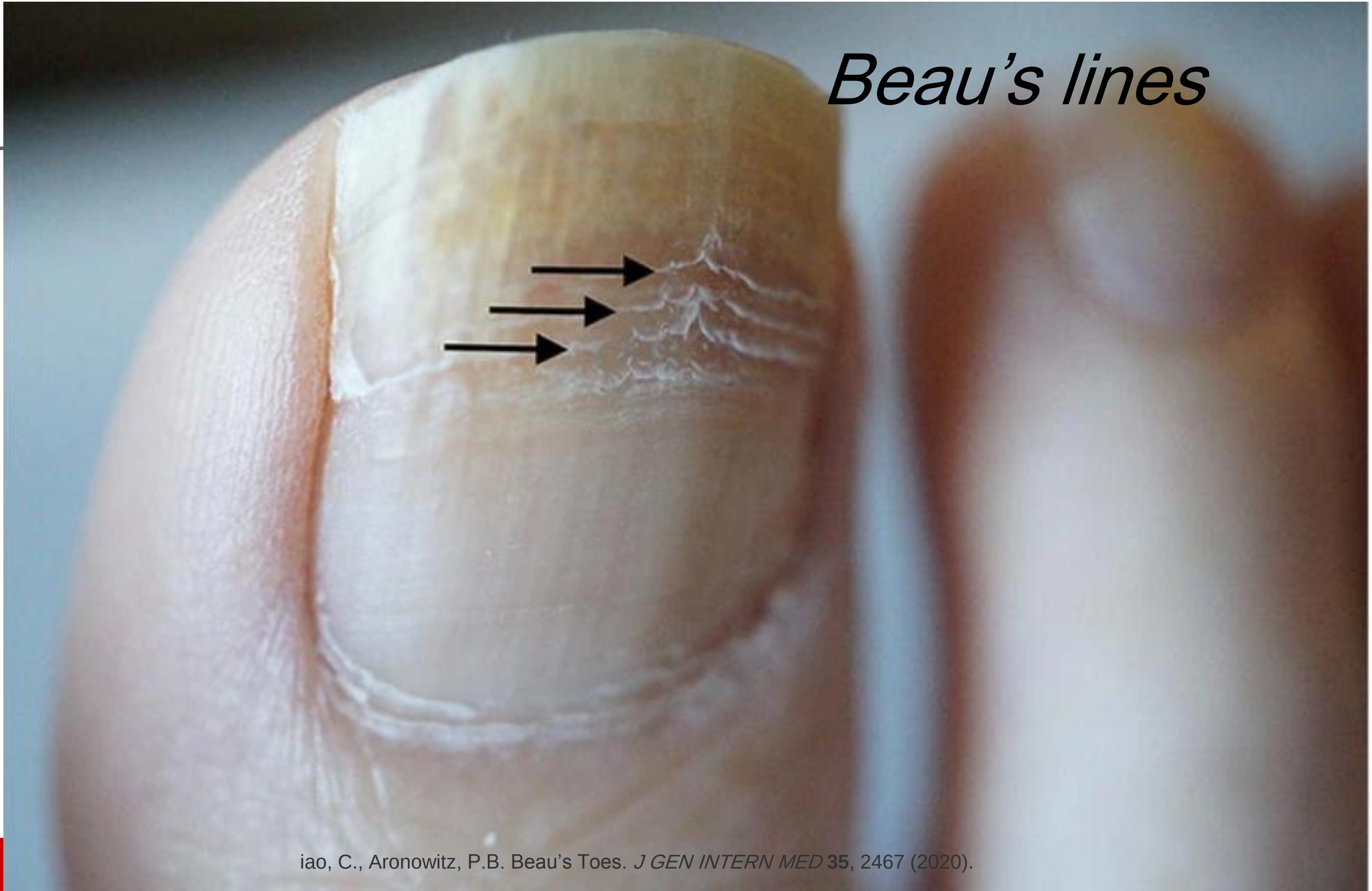
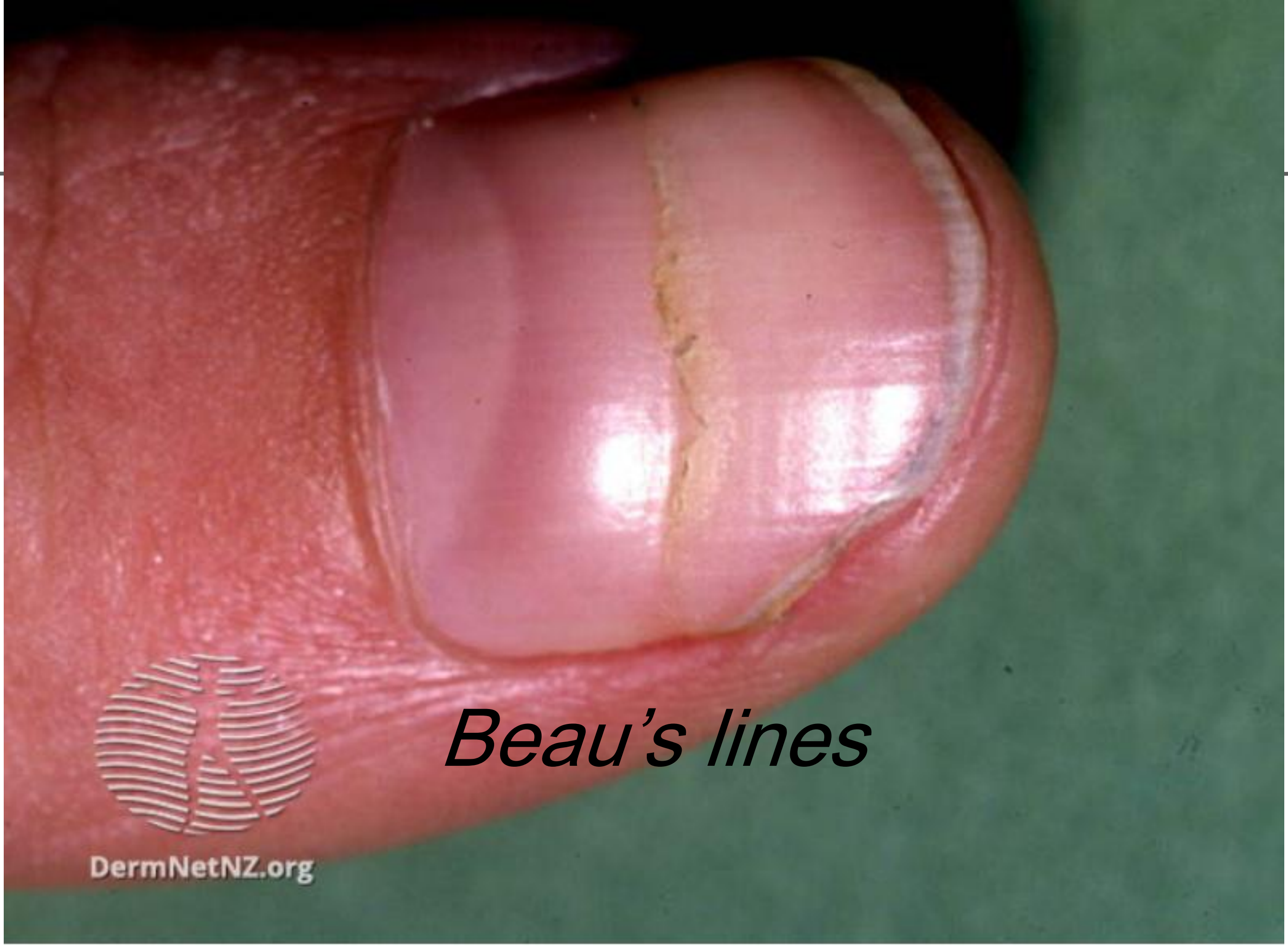
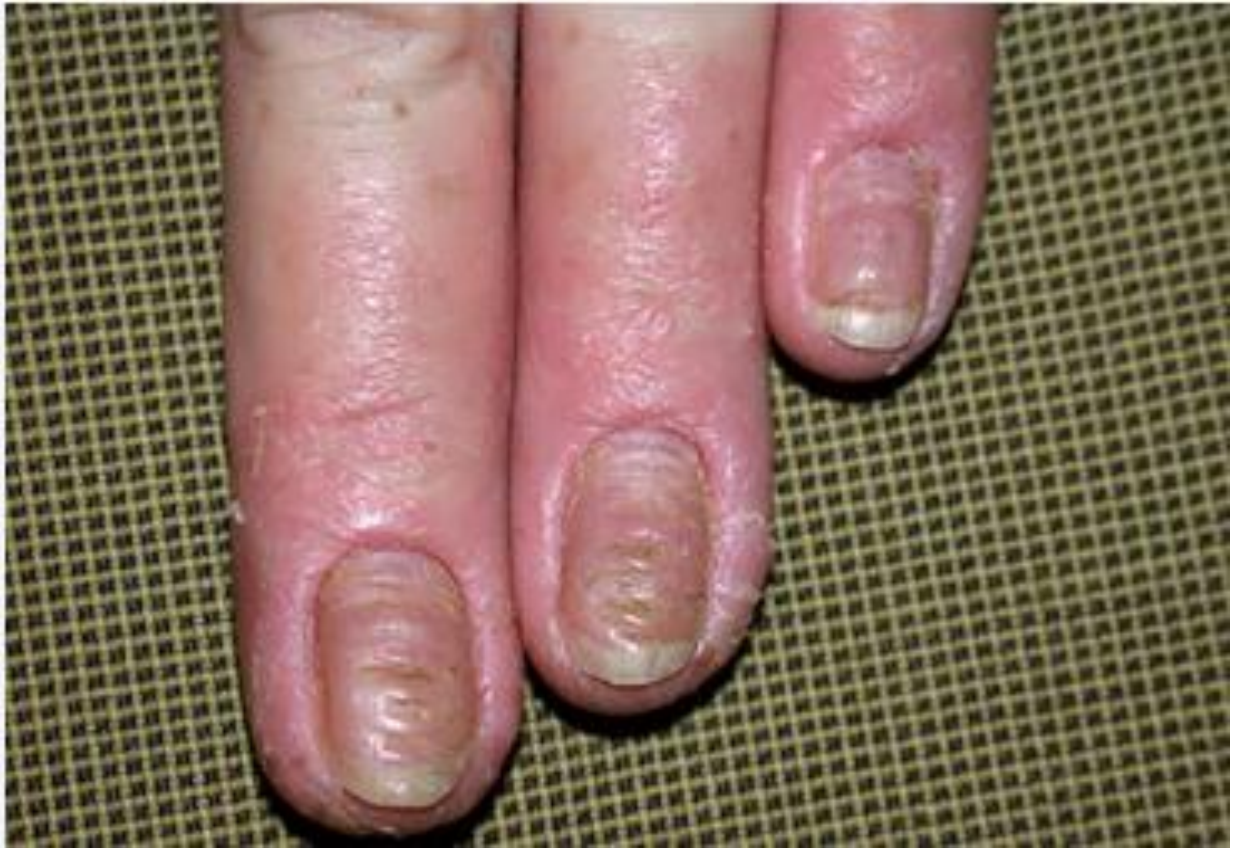
Beau’s lines



iao, C., Aronowitz, P.B. Beau’s Toes. *J GEN INTERN MED* **35**, 2467 (2020).


DermNetNZ.org

Beau’s lines



Beau’s lines

A Text Atlas of Nail Disorders Techniques in Investigation, Edited Robert Baran et al. third edition

1.Beau’s lines

- Transverse, band-like depressions extending from one lateral edge of the nail to the other,
- and affecting all nails at corresponding levels

- Beau’s lines are caused by temporary disruption in the growth of proximal nail matrix.

- Most often due to trauma or dermatological disorder (manicures, onychotillomania) or dermatologic disease of the proximal nail fold (e.g. eczema, chronic paronychia).
- Involvement of multiple digits suggests a systemic cause

- The distal limit of the furrow, if abrupt, indicates a sudden attack of disease; if sloping, a more protracted onset.

If the disease can completely inhibit the activity of the matrix for 1–2 weeks or longer, the transverse depression will result in total division of the nail plate, a defect known as ‘onychomadesis’

Table 3.2 Causes of transverse grooves

Common

- High fever
- Postnatal
- Menstrual cycle (multiple grooves), dysmenorrhoea
- Measles
- Trauma
- Chronic paronychia
- Local inflammation
- Chronic eczema

Uncommon

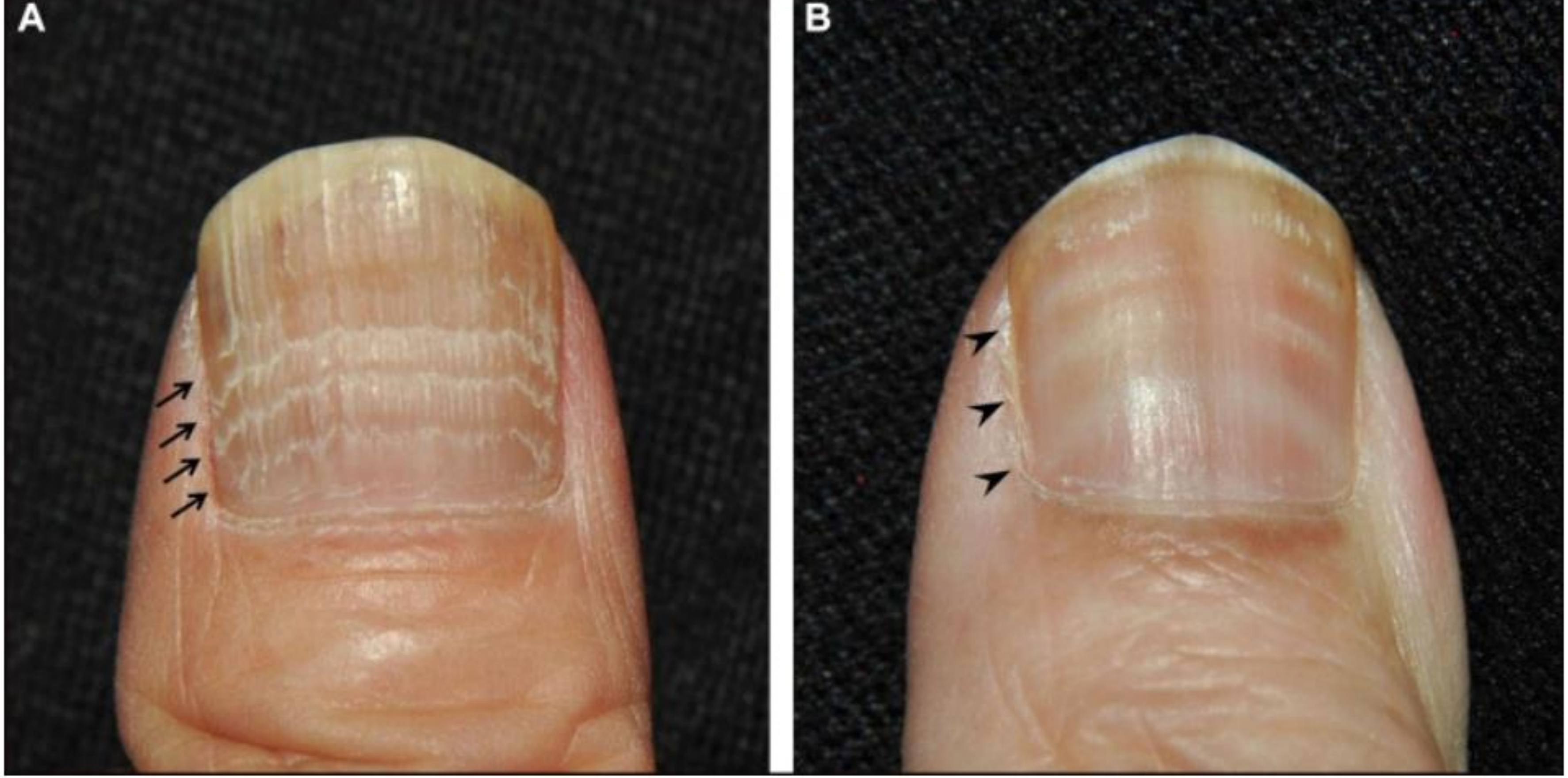
Kawasaki syndrome

Stevens-Johnson syndrome
Cytotoxic drugs
Acrodermatitis enteropathica and zinc deficiency
Hypoparathyroidism
Syphilis
Mouth, hand and foot disease
Radiation
Carpal tunnel syndrome

Differential diagnosis

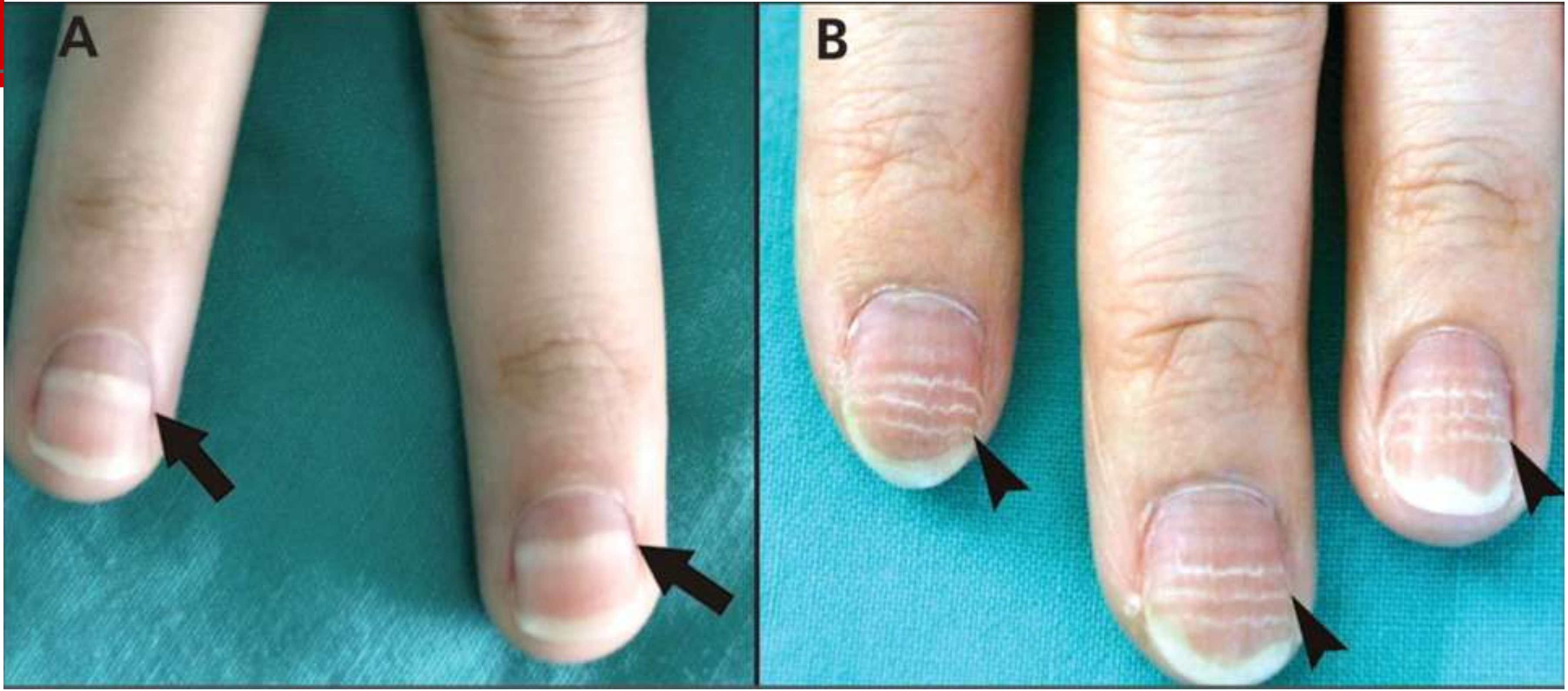
- Mees’ lines (true leukonychia)
- Muehrcke’s lines (apparent leukonychia)
- melanonychia

Palpable depression and non-blanchability differentiate Beau’s lines from Mees’ lines, other differential are Muehrcke’s lines and melanonychia



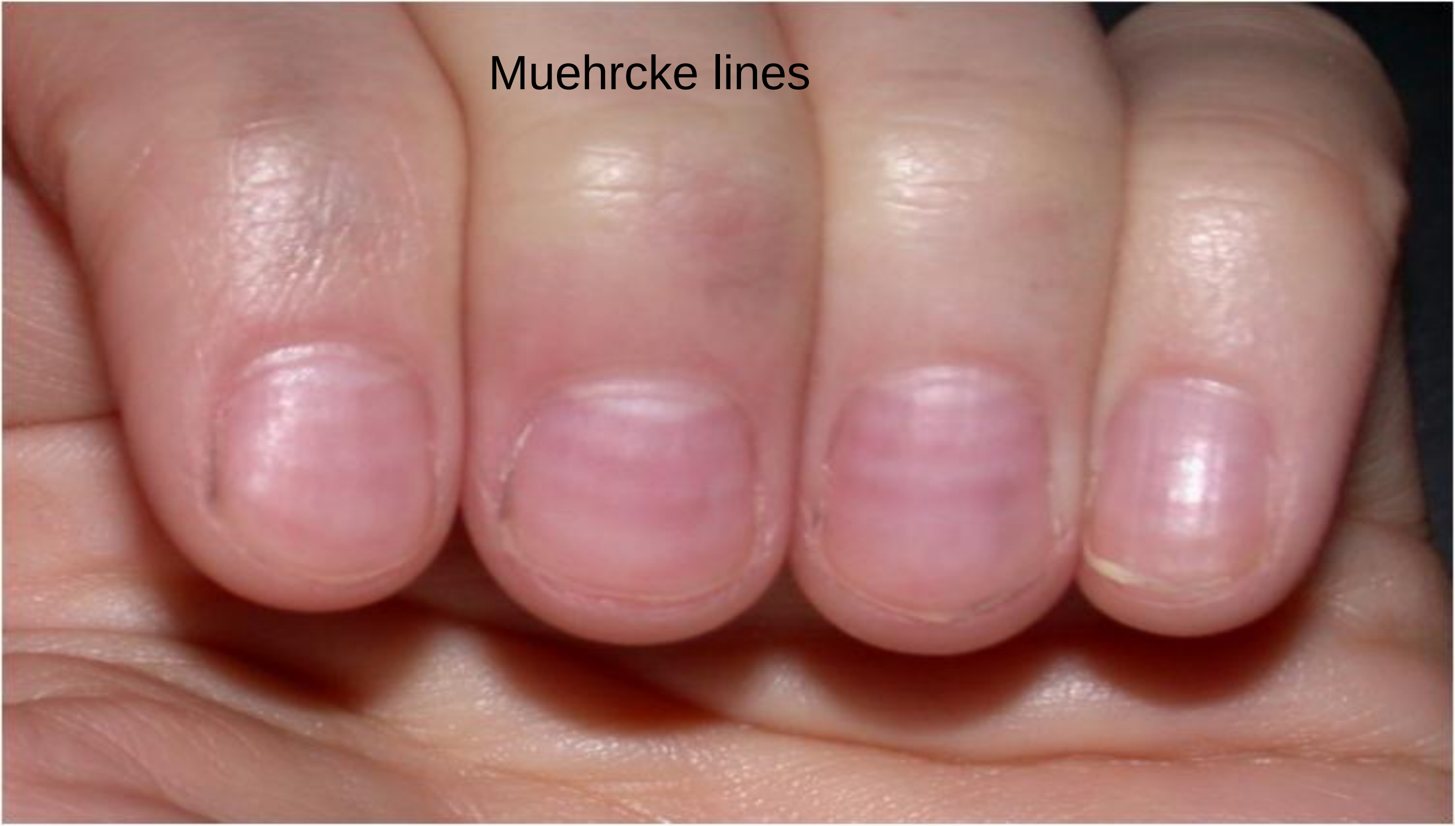
Transverse lines of the fingernails. (A) Beau's lines (*arrows*) of the thumbnail. (B) Mees' lines (*arrowheads*) of the thumbnail.

Song, Ik-Chan, and Hyo Jin Lee. "Transverse Lines of the Nails." *The American Journal of Medicine* 130.6 (2017): e259-e260



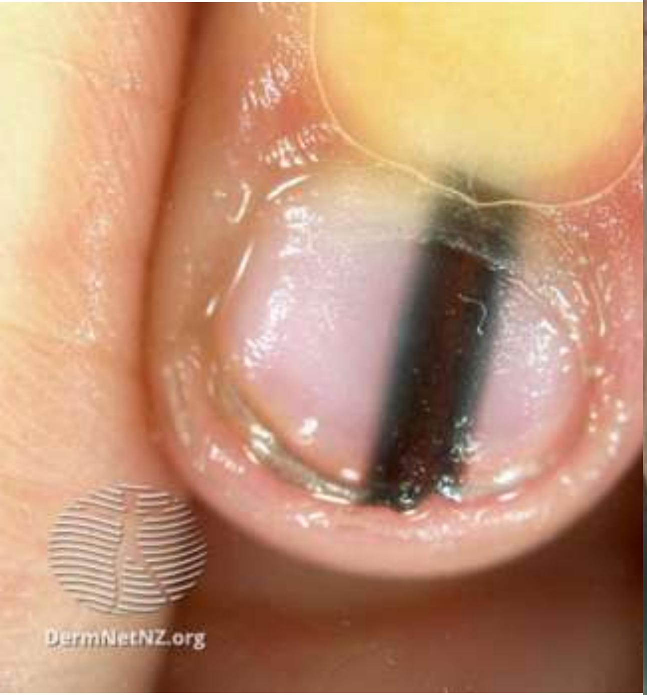
(A) Mees lines (arrows) in the fingernails of a 27-year-old woman. (B) Beau lines (arrowheads) in the fingernails of a 41-year-old woman.

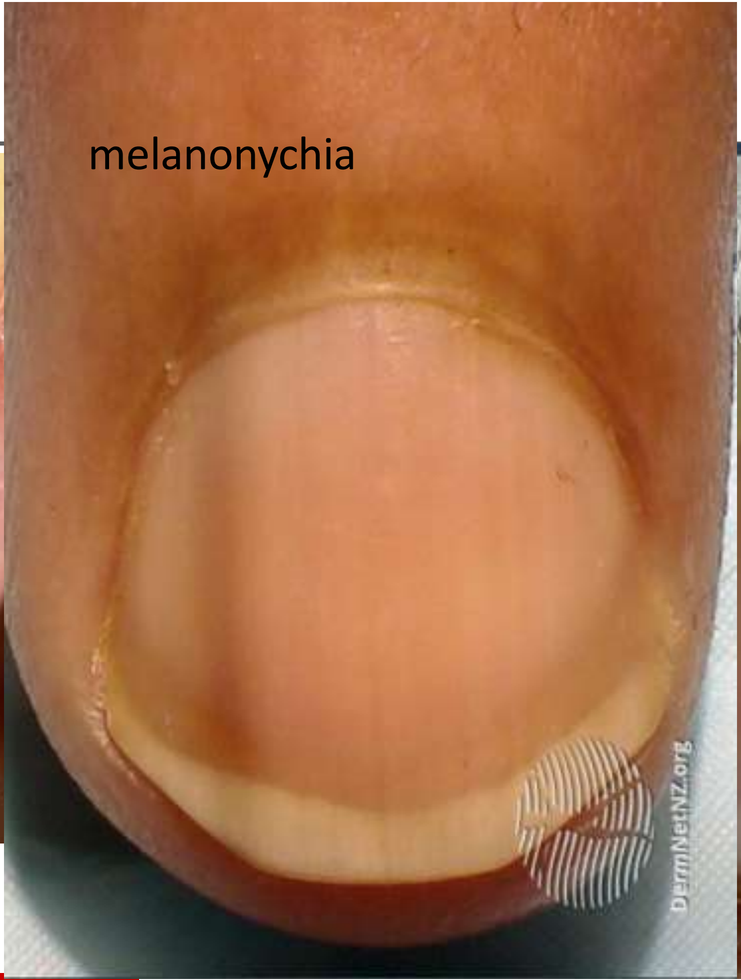
Huang, Tzu-Chuan, and Tsu-Yi Chao. "Mees lines and Beau lines after chemotherapy." *CMAJ* 182.3 (2010): E149-E149.

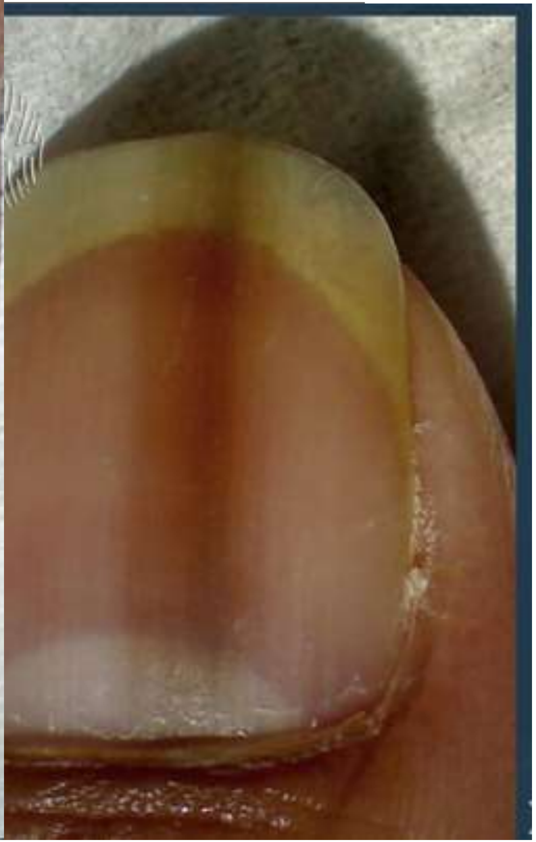


Muehrcke lines

Muehrcke lines are paired lines that fade when you press on them. Mees' lines are single lines that won't fade with pressure melanonychia


DermNetNZ.org

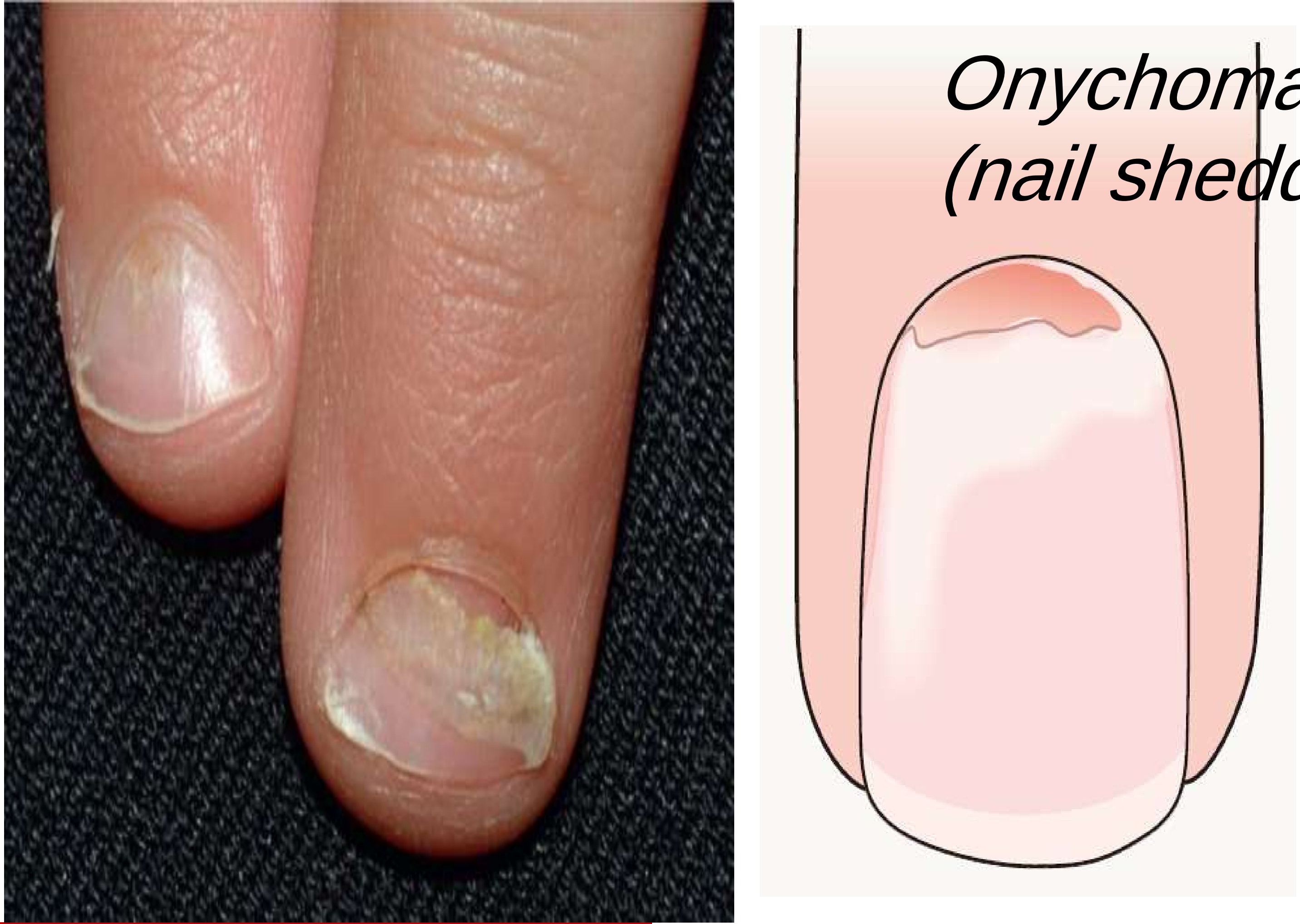

DermNetNZ.org



Treatment

There isn't a treatment for Beau's lines. But treating the underlying cause can keep them from coming back once they grow out.

Onychomadesis (nail shedding)



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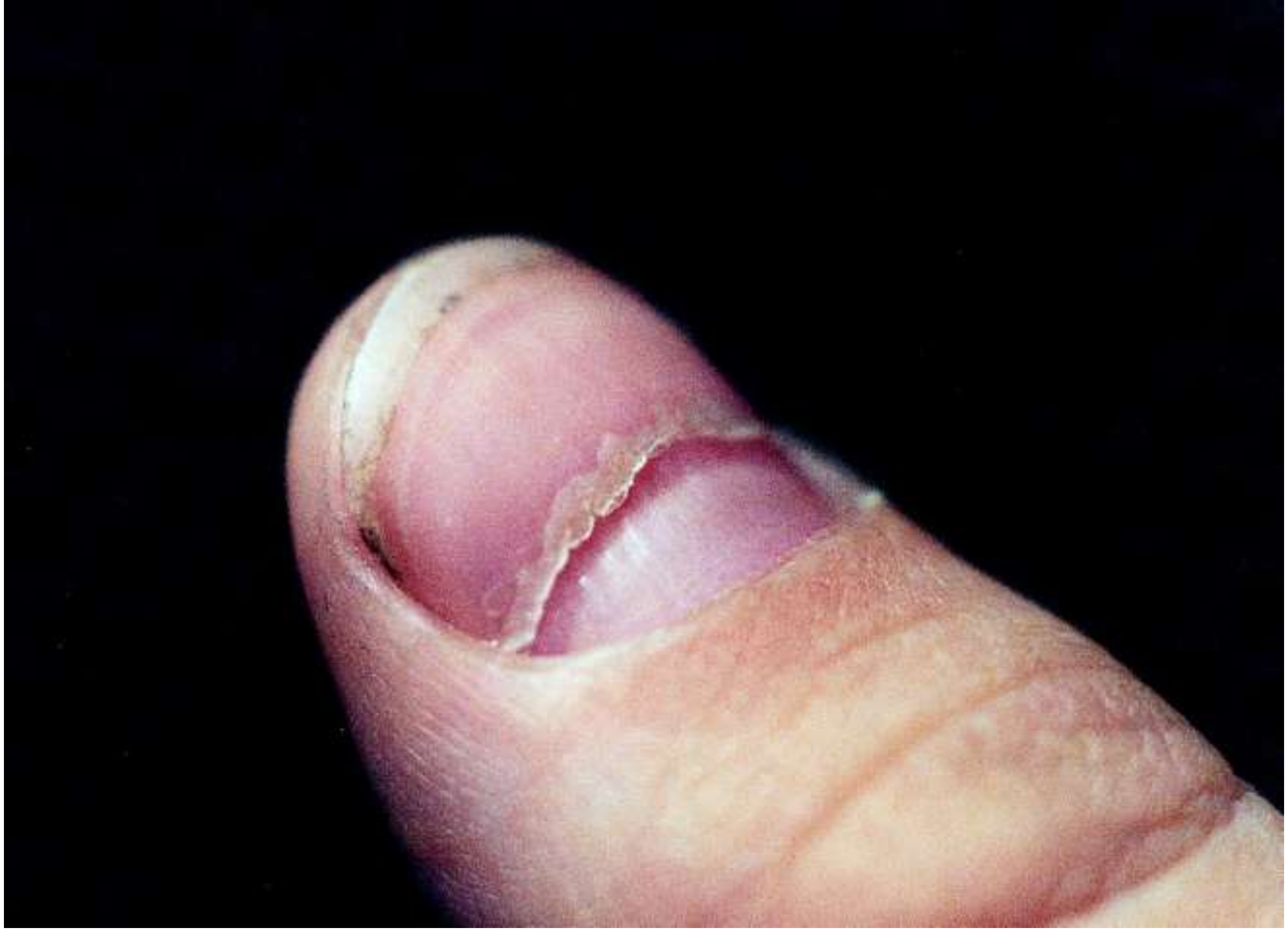
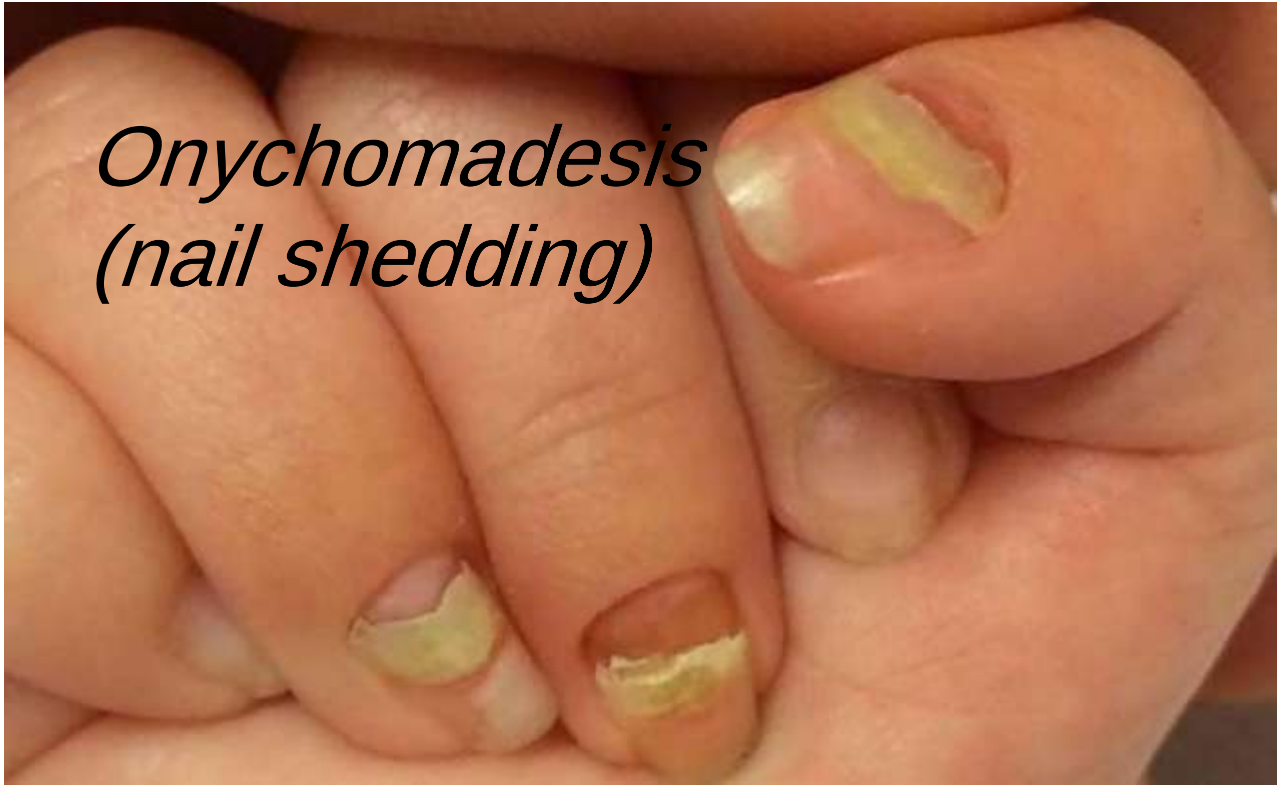
Onychomadesis



Ciastko, Allen R. "Onychomadesis and Kawasaki disease." *Cmaj* 166.8 (2002): 1069-1069.



Onychomadesis (nail shedding)

Bassi A, Greco A, de Martino M. Arch Dis Child Fetal Neonatal Ed

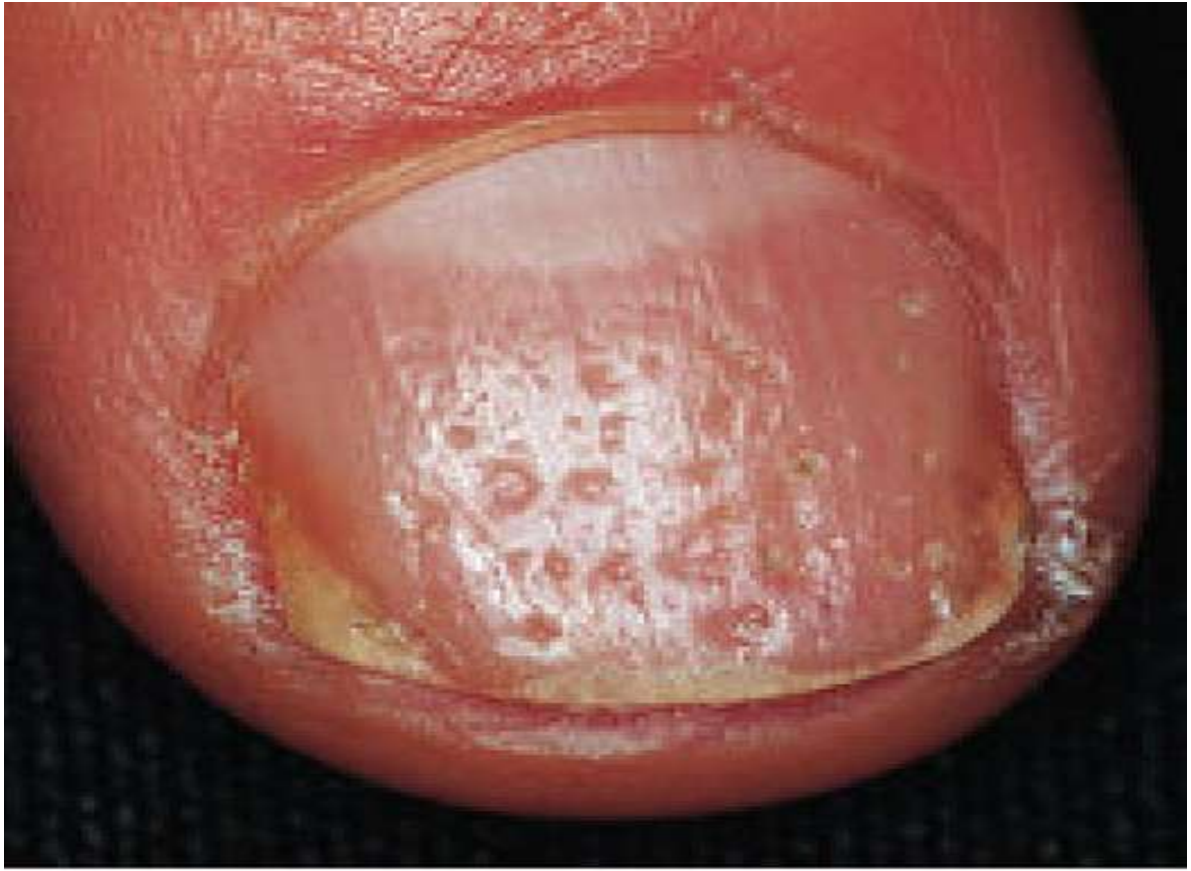
2.Onychomadesis (nail shedding)

Proximal detachment of the nail that is most often due to trauma

Similar involvement of multiple digits suggests a systemic cause

Table 4.2 Causes and associations of nail shedding

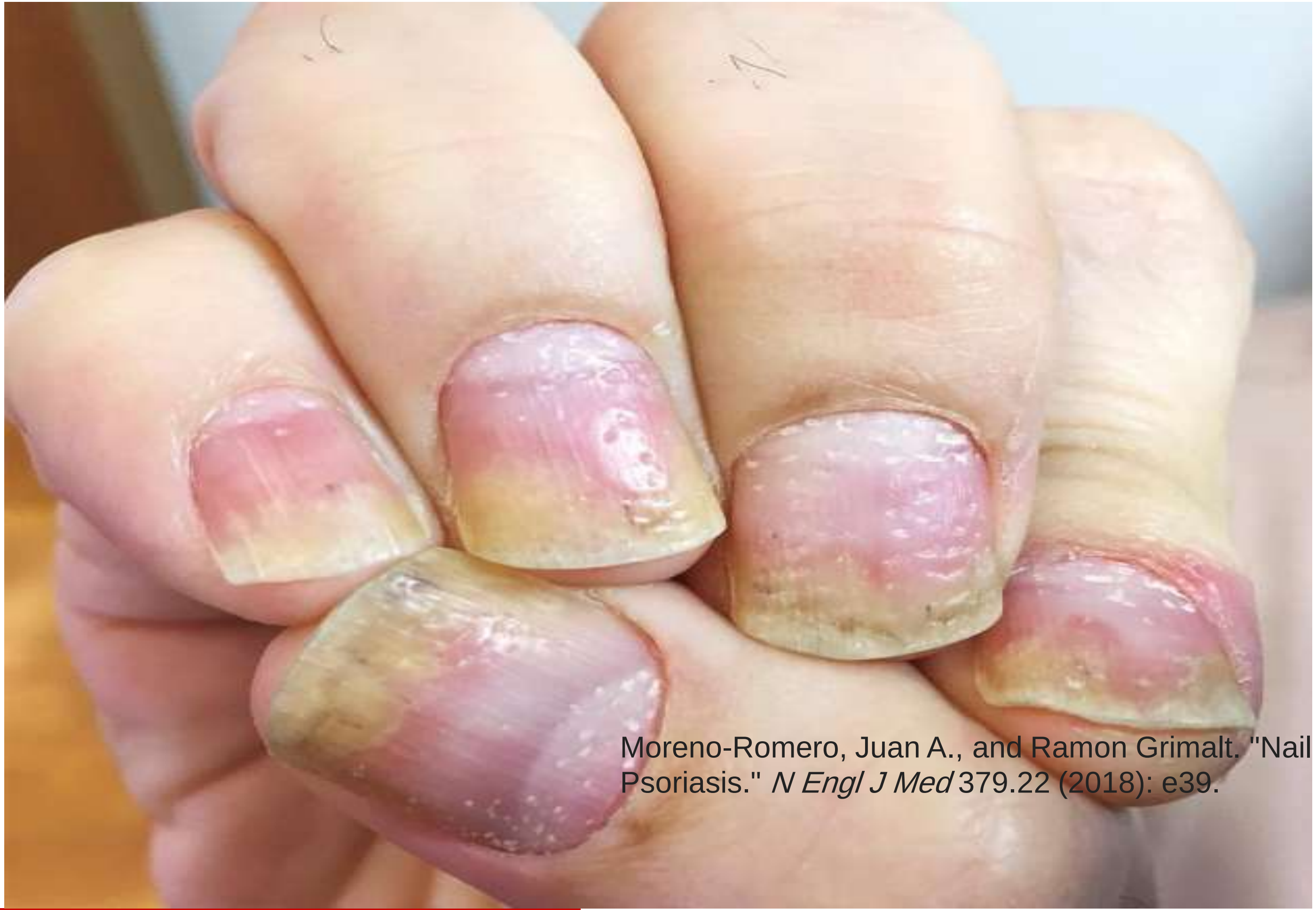
- Local inflammation, e.g. acute paronychia (Figures 4.7–4.12)
- Kawasaki's syndrome
- Fever or systemic upsets
- Syphilis
- Bullous dermatoses, e.g. pemphigus
- Stevens-Johnson syndrome
- Toxic epidermal necrolysis (Lyell's syndrome) (Figures 4.7–4.9)
- Drugs
  - Cytotoxics
  - Antibiotics
  - Retinoids
- Keratosis punctata
- Local trauma
- X-irradiation
- Acrodermatitis enteropathica
- Hypoparathyroidism with amelogenesis imperfecta
- Yellow nail syndrome



Pitting

A Text Atlas of Nail Disorders Techniques in Investigation, Edited Robert Baran et al. third edition

Pitting



Moreno-Romero, Juan A., and Ramon Grimalt. "Nail Pitting in Psoriasis." *N Engl J Med* 379.22 (2018): e39.

3. Pitting

Pitting describes the presence of punctate depressions of the nail plate surface that can have an irregular outline

Pits are due to foci of abnormal keratinization of the proximal nail matrix which result in clusters of parakeratotic cells within the dorsal nail plate.

In the proximal plate, these clusters appear as nontransparent collections of scale. Over time, shedding of the parakeratotic cells leads to the appearance of pits. These pits migrate distally with nail growth.

Table 3.3 Causes of pitting

Common

Psoriasis (Figures 3.26, 3.27, 3.29, 3.30)
Alopecia areata (Figure 3.31)
Eczema
Occupational trauma
Parakeratosis pustulosa

Uncommon

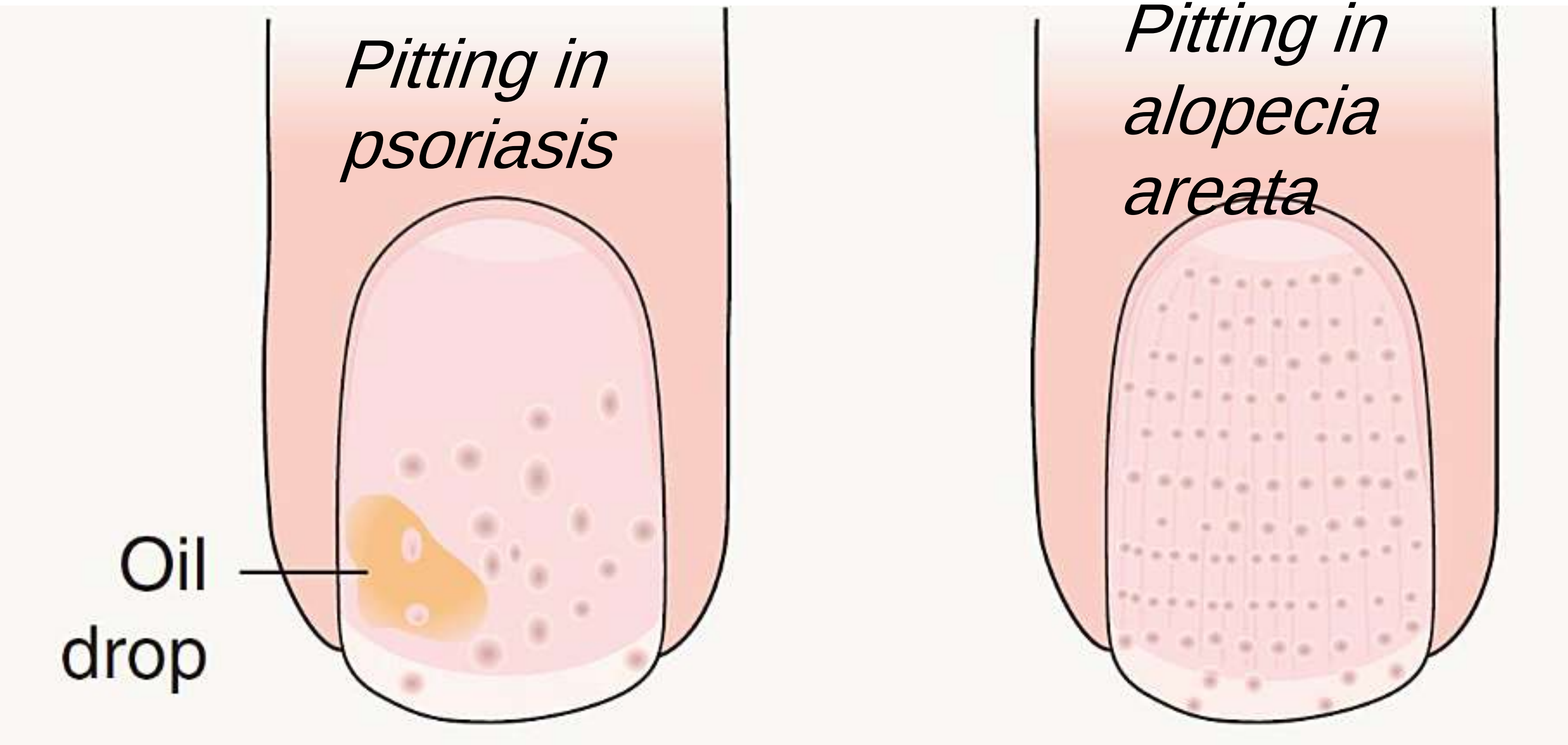
Normal
Pityriasis rosea
Secondary syphilis

Table 3.3 Causes of pitting

Sarcoidosis
Reiter’s syndrome
Lichen planus

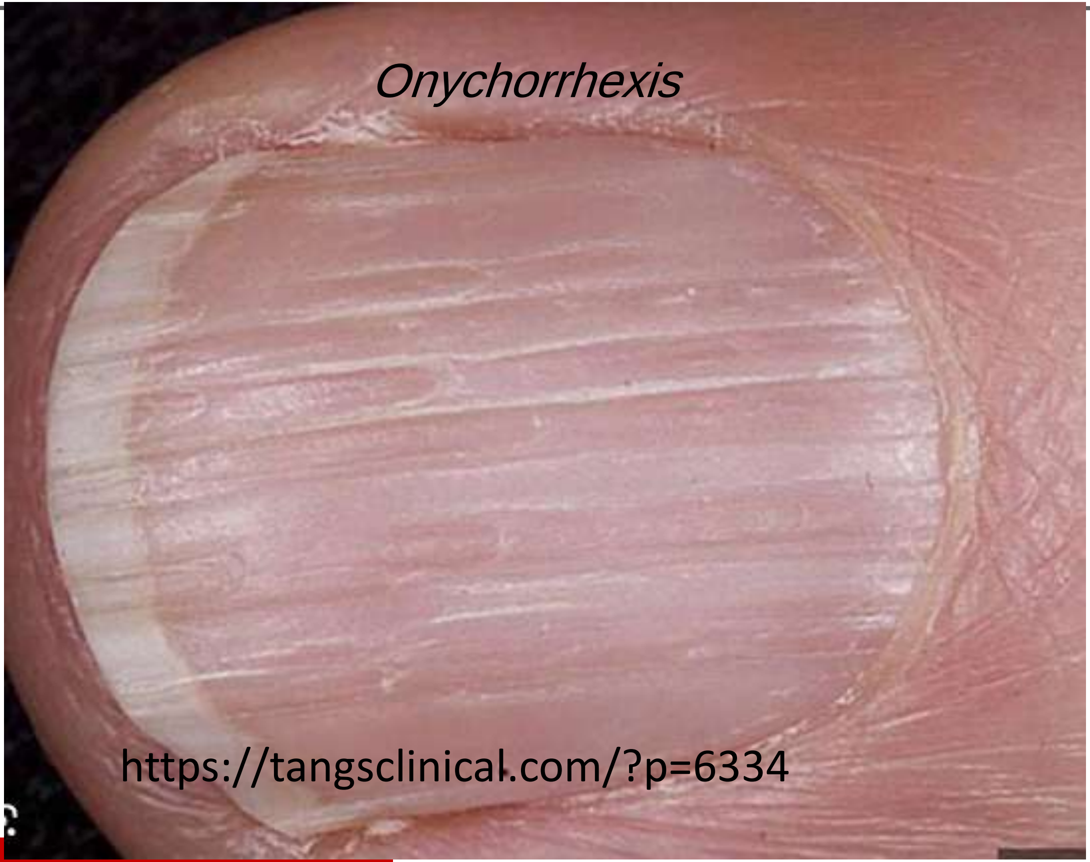
Large, deep and irregular pits are common in psoriasis and eczema, Small, superficial and regular pits are typical of alopecia areata

An isolated pit is not diagnostic and may be dye to minor trauma



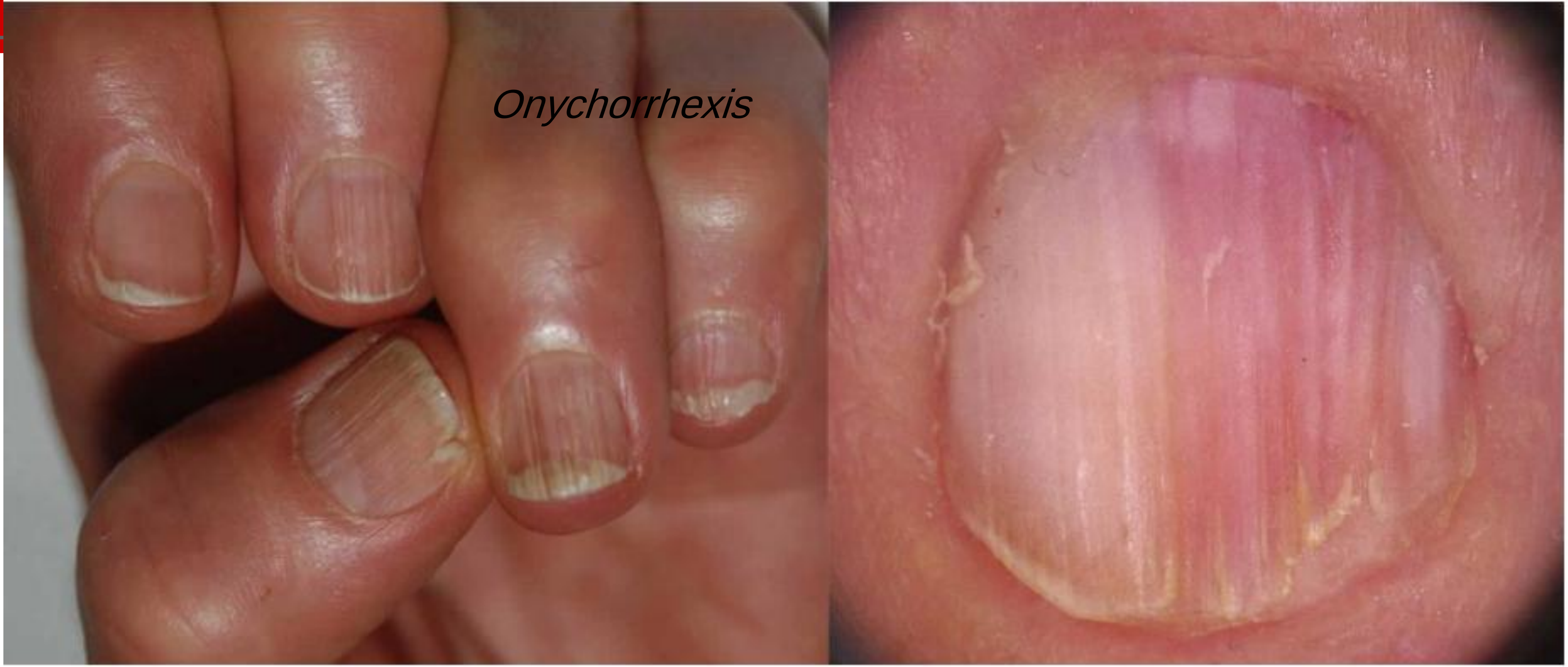
Pitting in psoriasis

Pitting in alopecia areata

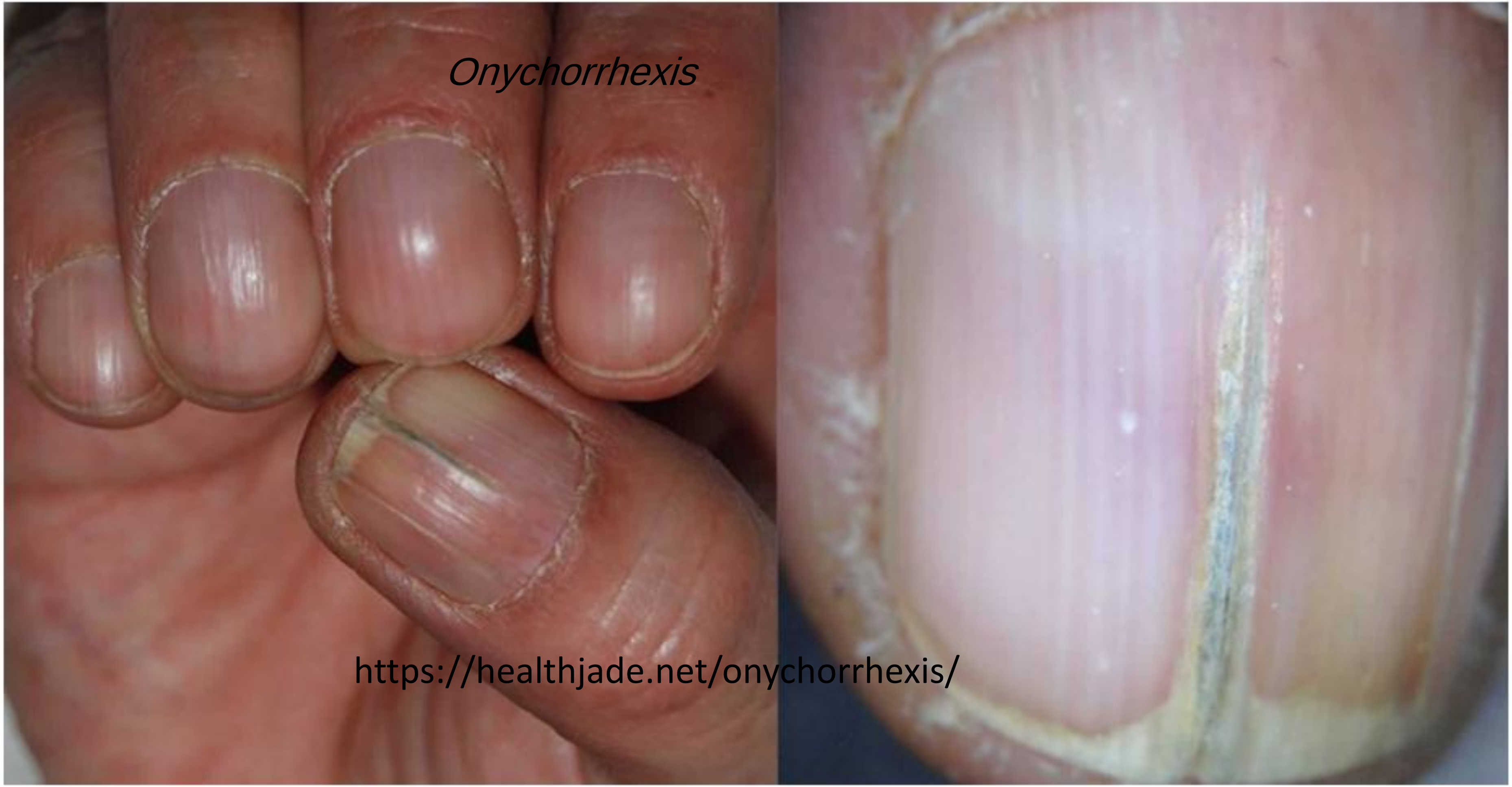


Onychorrhexis

https://tangsclinical.com/?p=6334



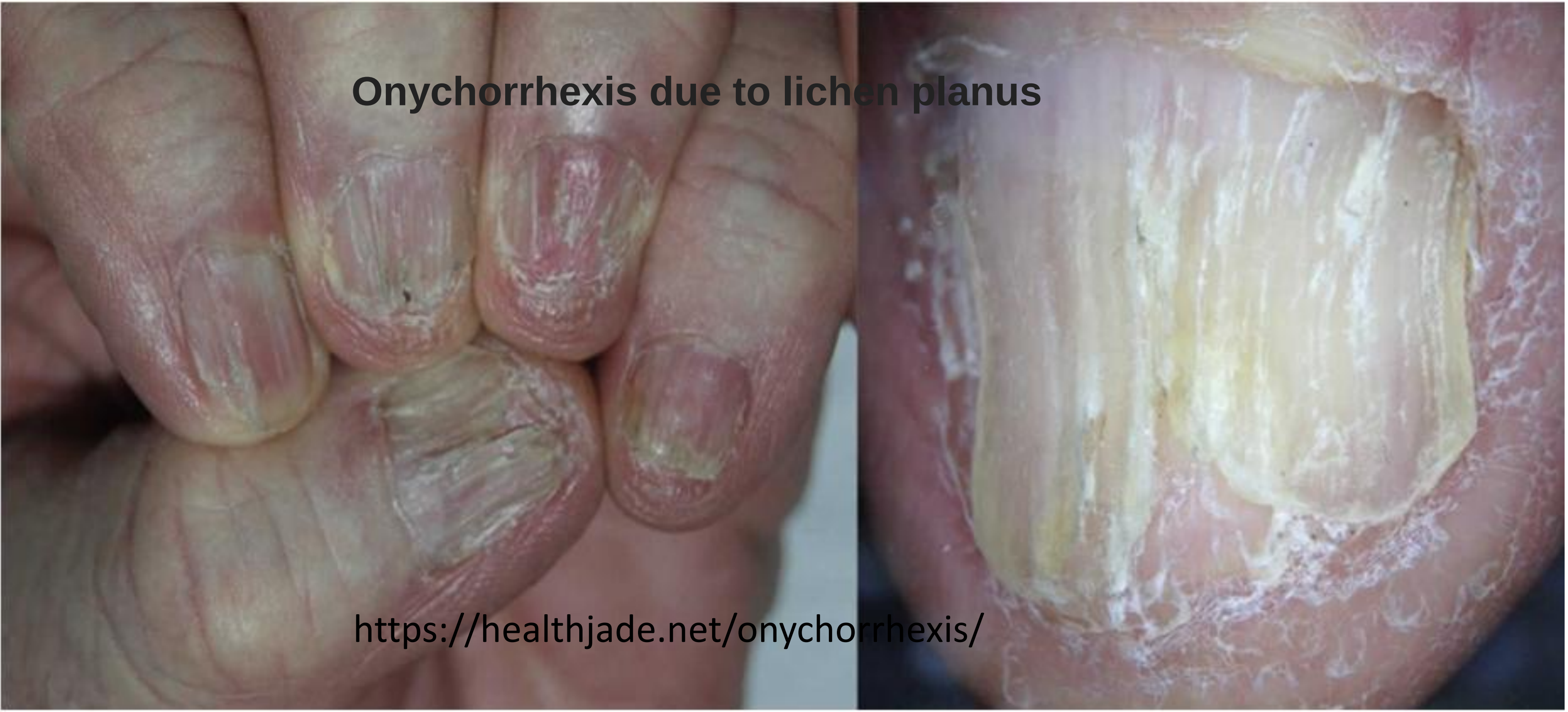
Onychorrhexis



Onychorrhexis

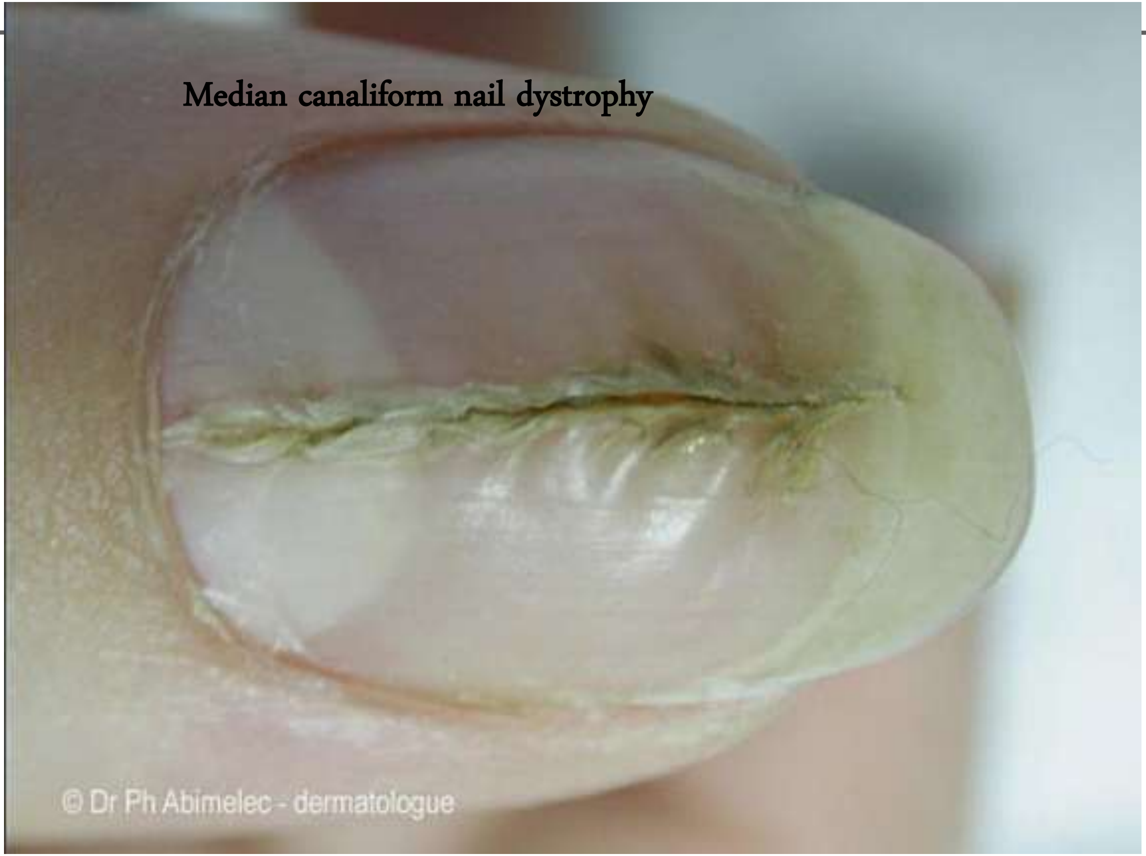
https://healthjade.net/onychorrhexis/

Onychorrhexis due to lichen planus



https://healthjade.net/onychorrhexis/

Median canaliform nail dystrophy



© Dr Ph Abimelec - dermatologue

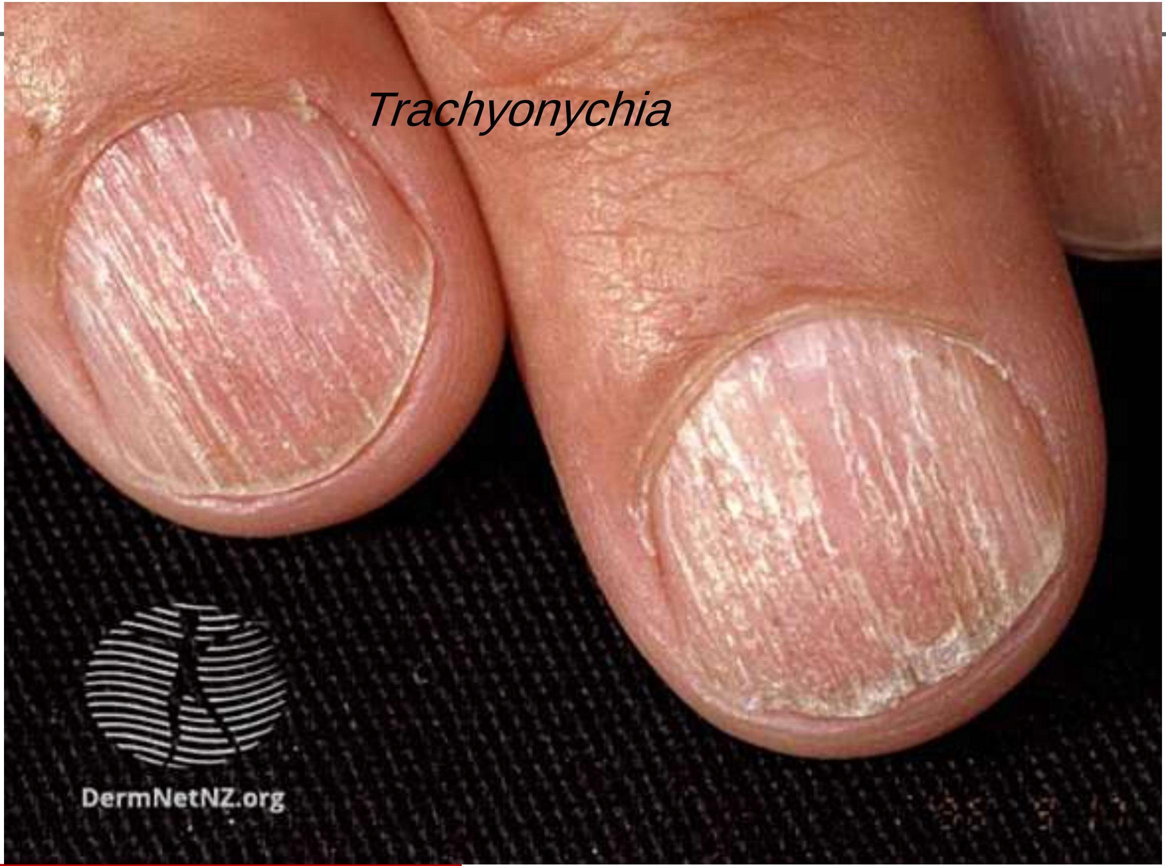
4. Onychorrhexis

Onychorrhexis is characterized by longitudinal ridging and fissuring of the nail plate. The depth of the ridges can vary as can the extent of involvement, Diseases commonly responsible for onychorrhexis include lichen planus, impaired vascular supply, trauma, and tumors that compress the nail matrix.

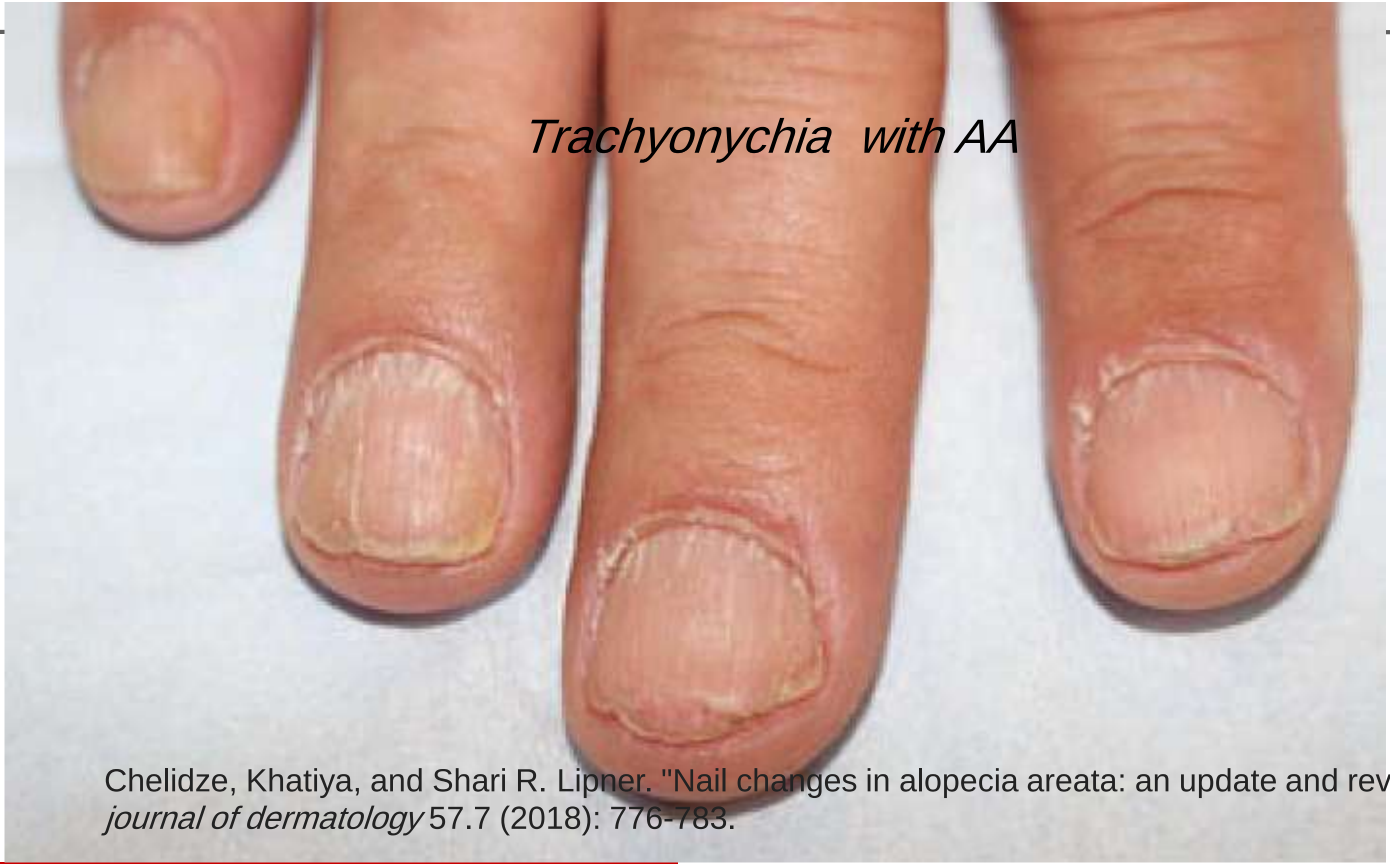
Table 3.1 Causes of longitudinal lines

Multiple	Normal; increase with age after early adulthood (Figures 3.1, 3.3, 3.4)
	With all causes of thin nail plates
	Lichen planus (Figure 3.5)
	Rheumatoid arthritis
	Graft-versus-host disease
	Psoriasis
	Darier's disease
	Poor circulation
	Collagen vascular diseases
	Radiation
	Frostbite
	Alopecia areata
	Nail-patella syndrome
	Systemic amyloidosis

Trachyonychia

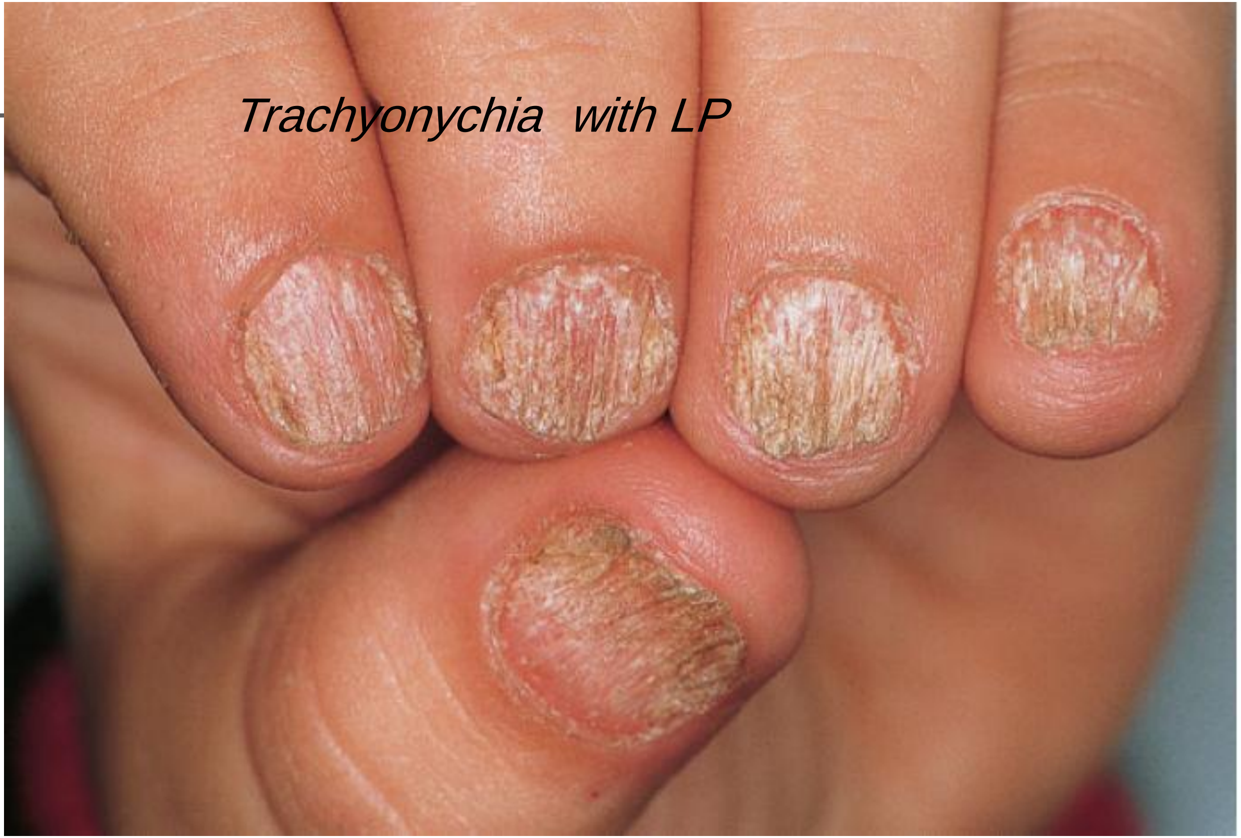

DermNetNZ.org

Trachyonychia with AA



Chelidze, Khatiya, and Shari R. Lipner. "Nail changes in alopecia areata: an update and review." *International journal of dermatology* 57.7 (2018): 776-783.

Trachyonychia with LP



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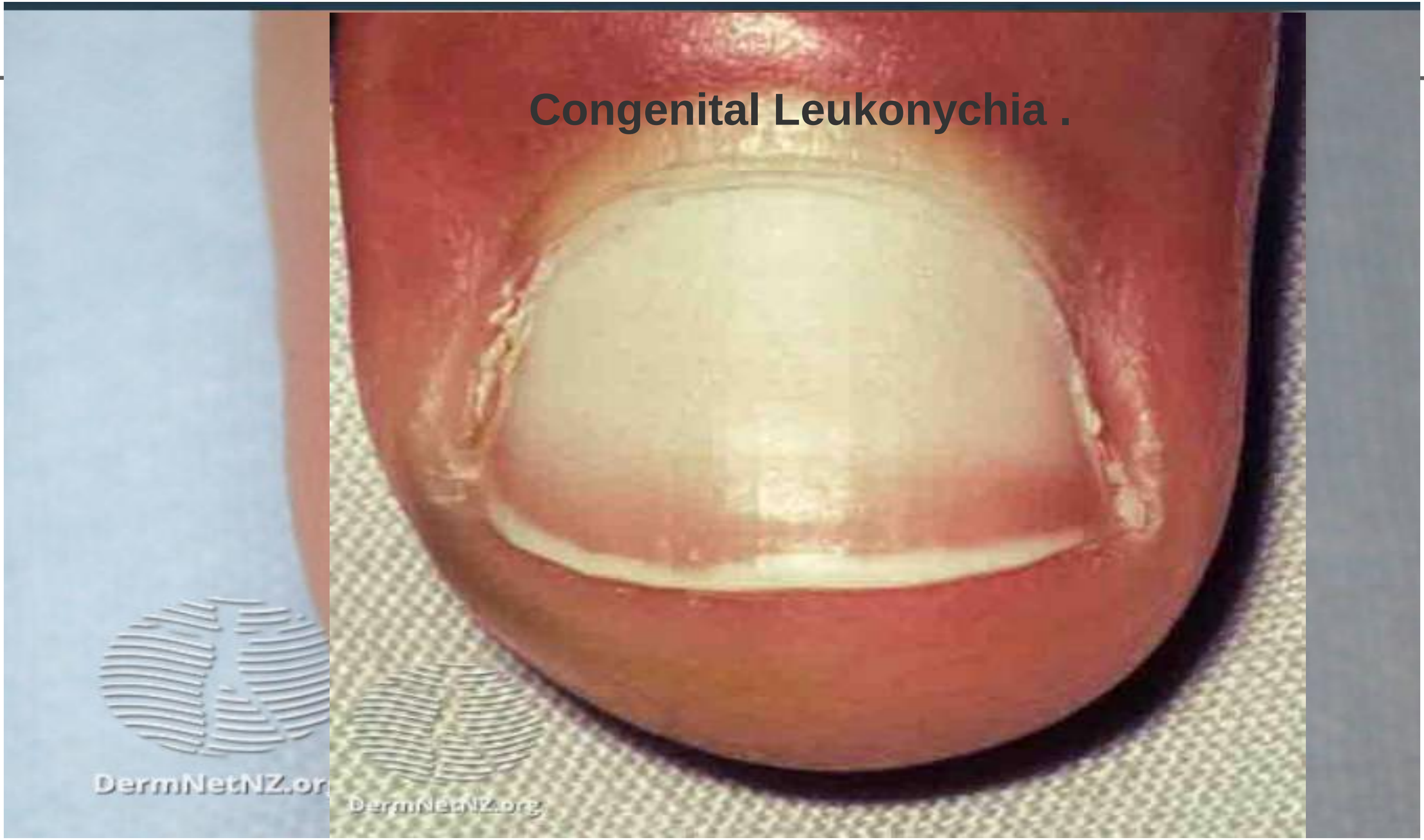
5. Trachyonychia (twenty-nail dystrophy, sandpapered nails

The term ‘twenty-nail dystrophy’ or trachyonychia describes a spectrum of nail plate surface abnormalities that result in nail roughness

Patients with trachyonychia can be divided into two main groups:
1 Trachyonychia and a past history or clinical evidence of alopecia areata.
2 Isolated nail involvement (idiopathic trachyonychia).

Congenital Leukonychia .


DermNetNZ.org
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6. leukonychia

White nails are the most common form of colour change. These can be divided into the following main types:

1 True leukonychia, in which the nail plate is involved.

2 Apparent leukonychia, with involvement of the subungual tissue. (Nail Signs Due to Nail Bed Disorders)

3 Pseudoleukonychia.

Table 7.1 Causes of leukonychia

Congenital and/or hereditary

Isolated
Acrokeratosis verruciformis (Hopf)
Associated with koilonychia (Figure 7.8)
LEOPARD syndrome (lentigines, electrocardiographic changes, ocular hypertelorism, pulmonary stenosis, abnormalities of genitalia, retarded growth, deafness)
Associated with deafness
Leukonychia totalis, multiple sebaceous cysts, renal calculi
Darier’s disease—usually linear and longitudinal

Acquired

Pseudoleukonychia

- Diffuse form of distal and lateral subungual onychomycosis
- Proximal white subungual onychomycosis, especially in AIDS patients
- Superficial white onychomycosis
- Keratin granulation (superficial friability from nail varnish)
- Psoriasis

Apparent leukonychia (Figure 7.9)

- Anaemia
- Cancer chemotherapeutic agents
- Cirrhosis (Terry's sign)
- Dyshidrosis
- Half-and-half nail (renal diseases) and distal crescent pigmentation
- Leprosy

Acquired

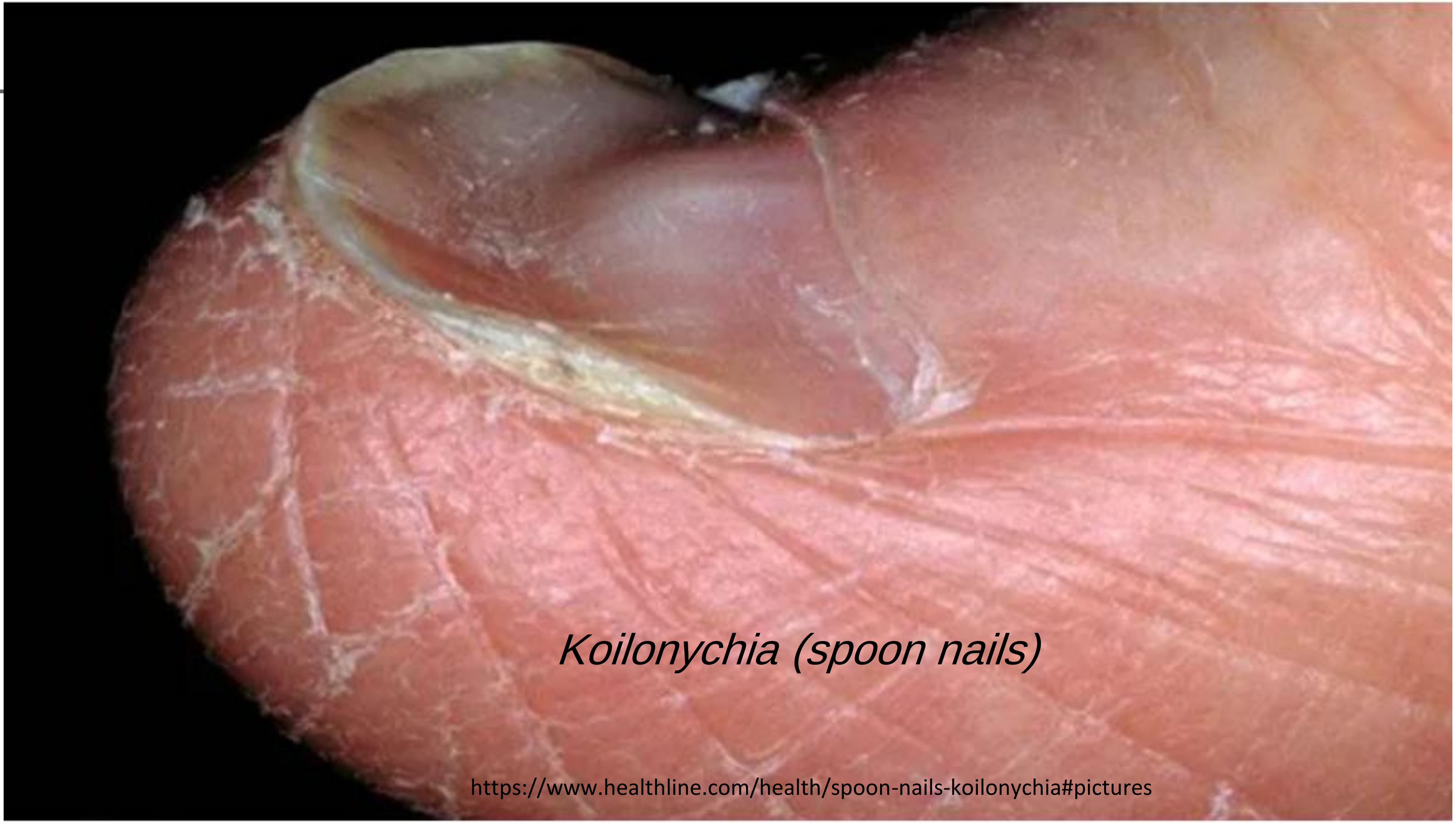
Muehrcke’s lines of hypoalbuminaemia
True leukonychia
Alkaline metabolic disease
Alopecia areata
Carcinoid tumours of the bronchus
Cardiac insufficiency
Cytotoxic and other drugs (emetine, pilocarpine, sulphonamide, cortisone)
Erythema multiforme
Exfoliative dermatitis
Fasting periods (e.g. in orthodox Jews and Muslims)
Fracture
Gout
Hodgkin’s disease

Acquired

Hypocalcaemia
Infectious diseases and infectious fevers
Intra-abdominal malignancies
Kidney transplant
Leprosy
Leuko-onycholysis paradentotica
Menstrual cycle
Myocardial infarction
Occupational
Pellagra
Peripheral neuropathy
Poisoning (antimony, arsenic, fluoride, lead, thallium)
Protein deficiency
Psoriasis
Psychotic episodes (acute)
Renal failure (acute or chronic)
Shock
Sickle cell anaemia

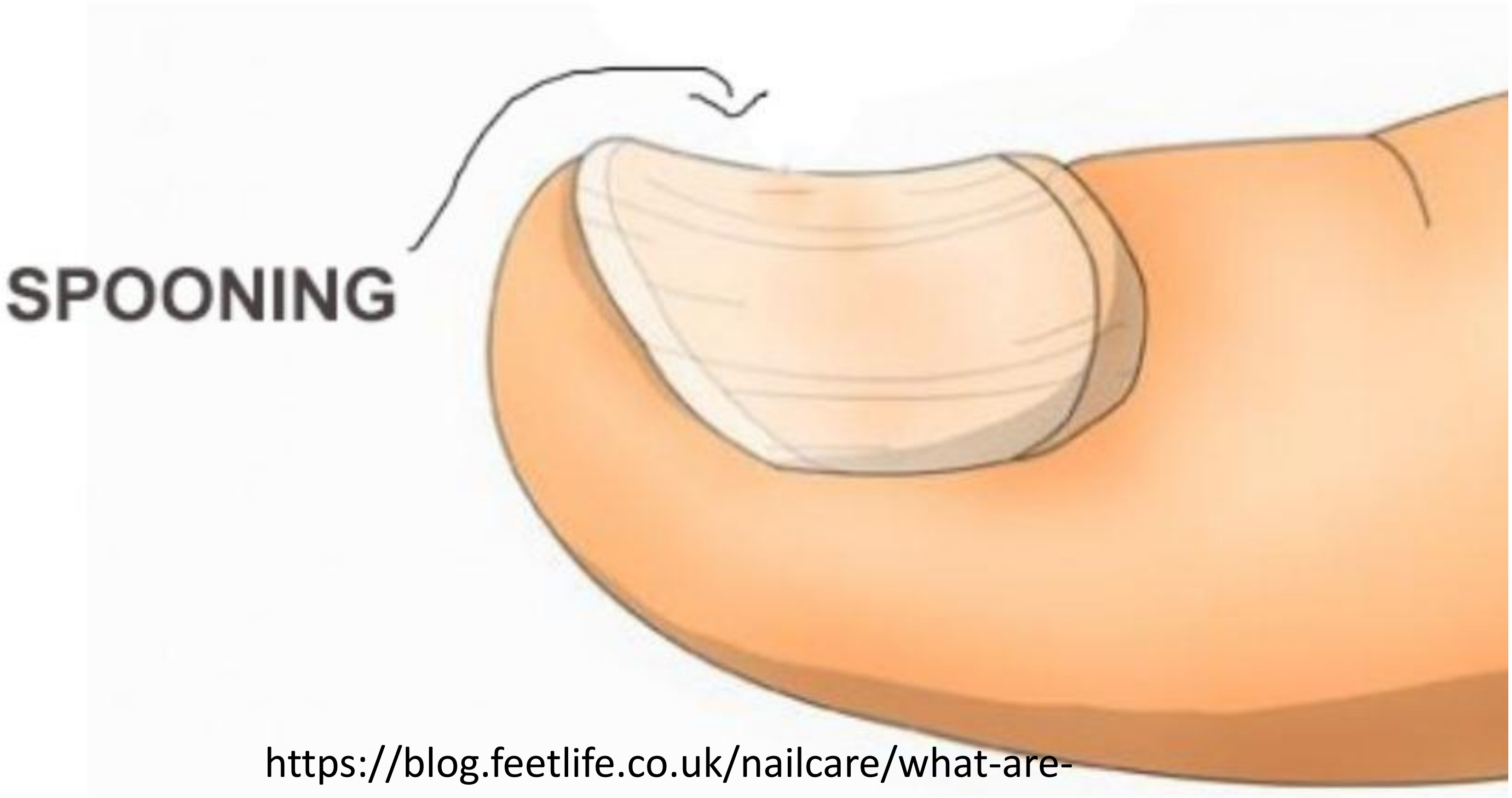
Acquired

Sickle cell anaemia
Surgery
Sympathetic leukonychia
Trauma (single or repeated)
Tumours (benign), cysts pressing on matrix
Ulcerative colitis
Zinc deficiency



Koilonychia (spoon nails)

https://www.healthline.com/health/spoon-nails-koilonychia#pictures



https://blog.feetlife.co.uk/nailcare/what-are-spoon-shaped-nails/

7.Koilonychia (spoon nails)

The nail plate is thinned and flattened with upward eversion of its lateral and distal edges, leading to a concave spoon-like shape

Table 2.2 Common causes of koilonychia

Physiological

- Early childhood (Figures 2.12–2.13)

Idiopathic

Congenital

- LEOPARD syndrome
- Ectodermal dysplasias (Figure 2.15)
- Trichothiodystrophy
- Nail-patella syndrome

Acquired

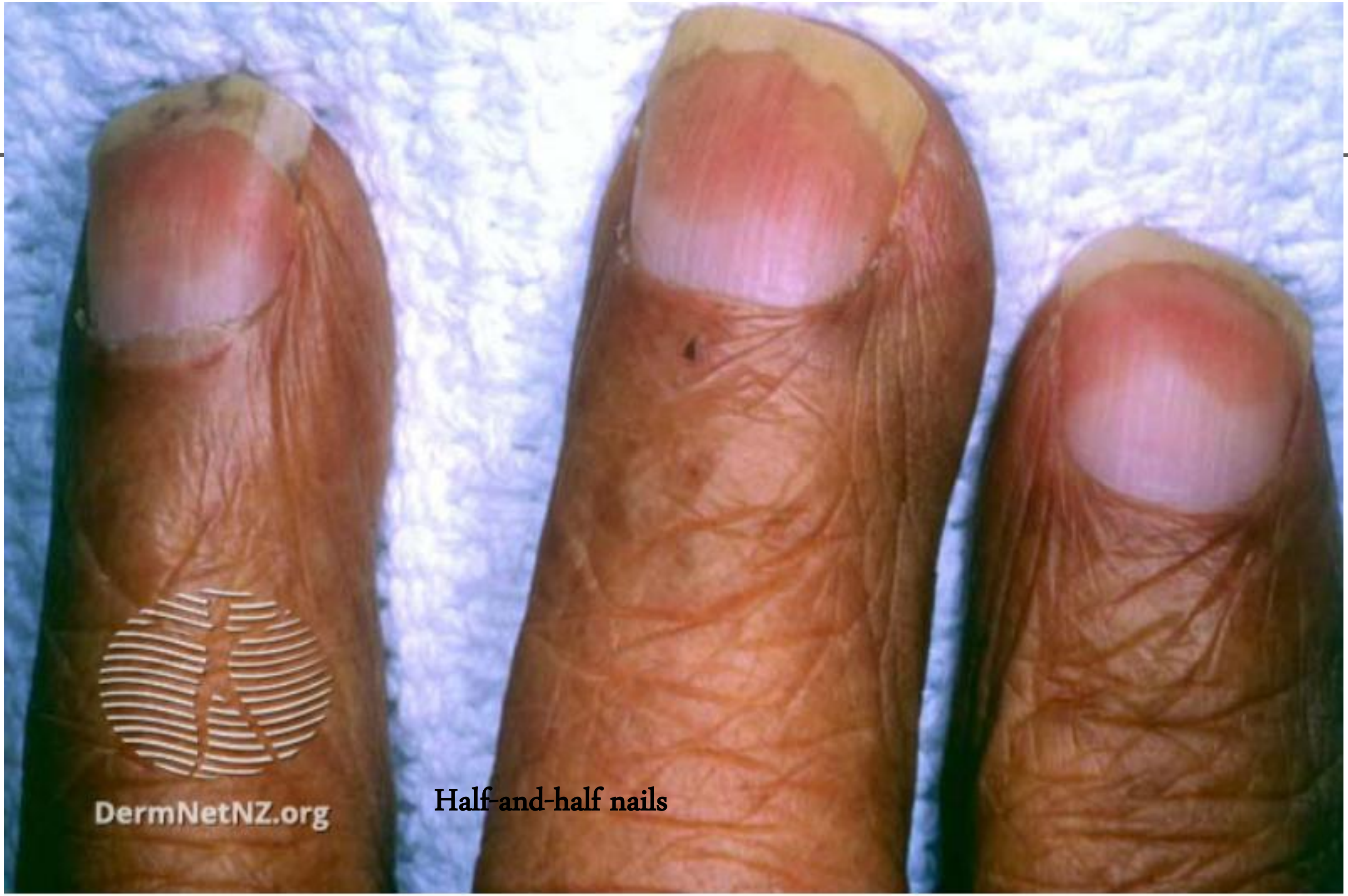
- Metabolic/endocrine
- iron deficiency
- acromegaly
- haemochromatosis
- porphyria
- renal dialysis/transplant
- thyroid disease
- Dermatoses

Table 2.2 Common causes of koilonychia

alopecia areata
Darier’s disease
lichen planus
psoriasis
Raynaud’s disease
Occupational
contact with oils, e.g. engineering industry
Infections
onychomycosis
syphilis
Traumatic

toes of rickshaw pullers
Carpal tunnel syndrome

Nail Signs Due to Nail Bed Disorders



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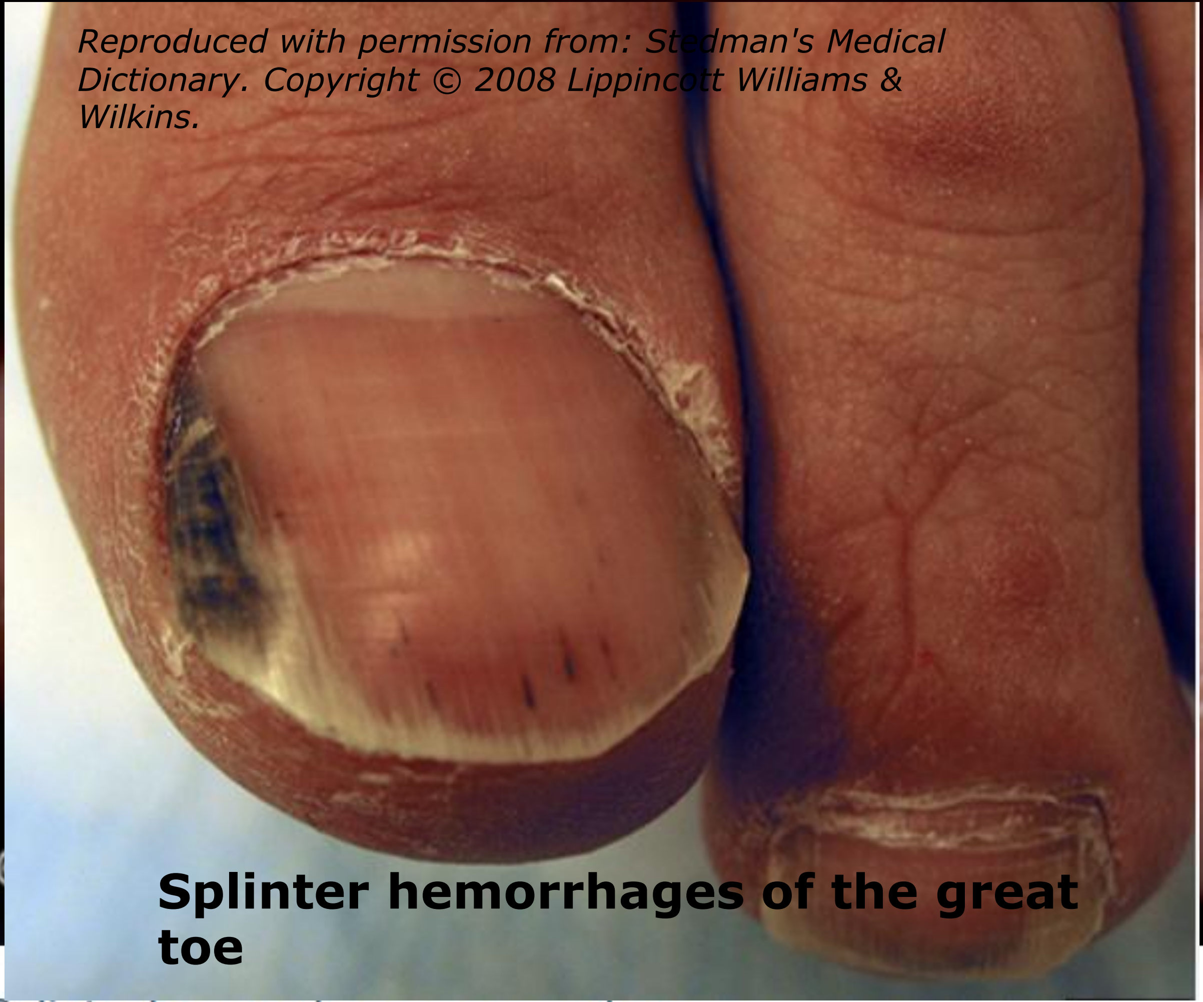
Half-and-half nails



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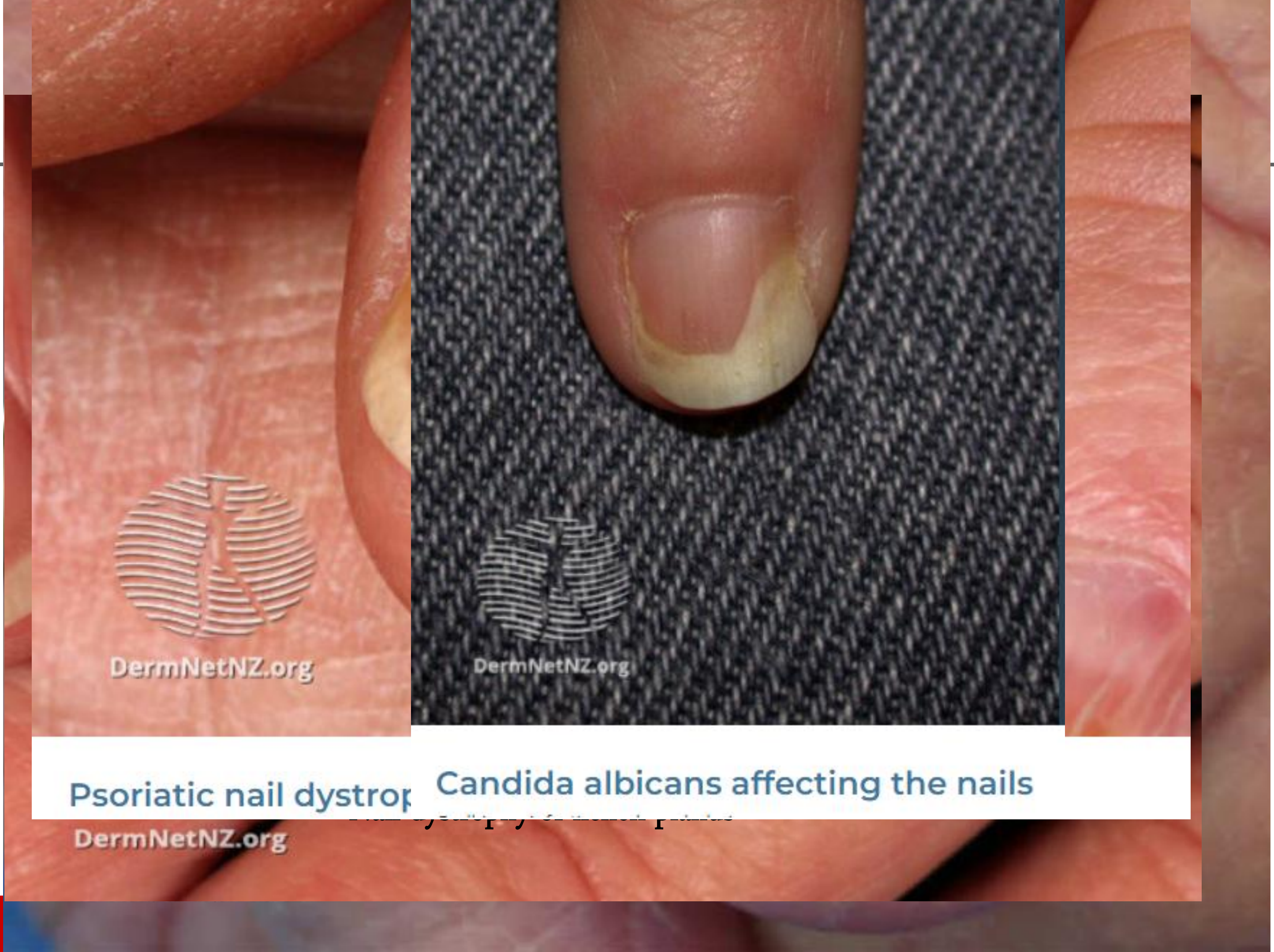
Splinter hemorrhages of the great toe

Splinter haemorrhages seen end on-on

1.Splinter hemorrhages

Splinter hemorrhages appear as one or more red–brown to purple–black, thin longitudinal lines in the distal portion of the nail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Psoriatic nail dystrop

Candida albicans affecting the nails

2.Onycholysis

The distal nail plate is detached from the nail bed and usually appears white because of the presence of air in the subungual space. If an exogenous pigment is present, the nail may appear yellow (fungi and exudate) to green–black (pyocyanin).

Diseases commonly responsible for onychorrhexis include : lichen planus, impaired vascular supply, trauma, and tumors that compress
the nail matrix. The differential diagnosis includes the common phenomenon of age-related ridging of the nail plate.

Table 4.1 Some causes of onycholysis

Idiopathic

Leuko-onycholysis paradentotica (Schuppli syndrome)
Of women (cosmetic use)

Systemic

Circulatory (e.g. lupus erythematosus)
Yellow nail syndrome
Endocrine (e.g. hypothyroidism, thyrotoxicosis)
Pregnancy
Syphilis
Iron deficiency anaemia
Carcinoma of the lung
Pellagra

Table 4.1 Some causes of onycholysis

Congenital and/or hereditary

Partial hereditary onycholysis

Pachyonychia congenita

Cutaneous diseases

Psoriasis, Reiter's disease, vesicular or bullous disease, lichen planus, alopecia areata, multicentric reticulohistiocytosis

Atopic dermatitis, contact dermatitis (accidental or occupational), mycosis fungoides, actinic reticuloid

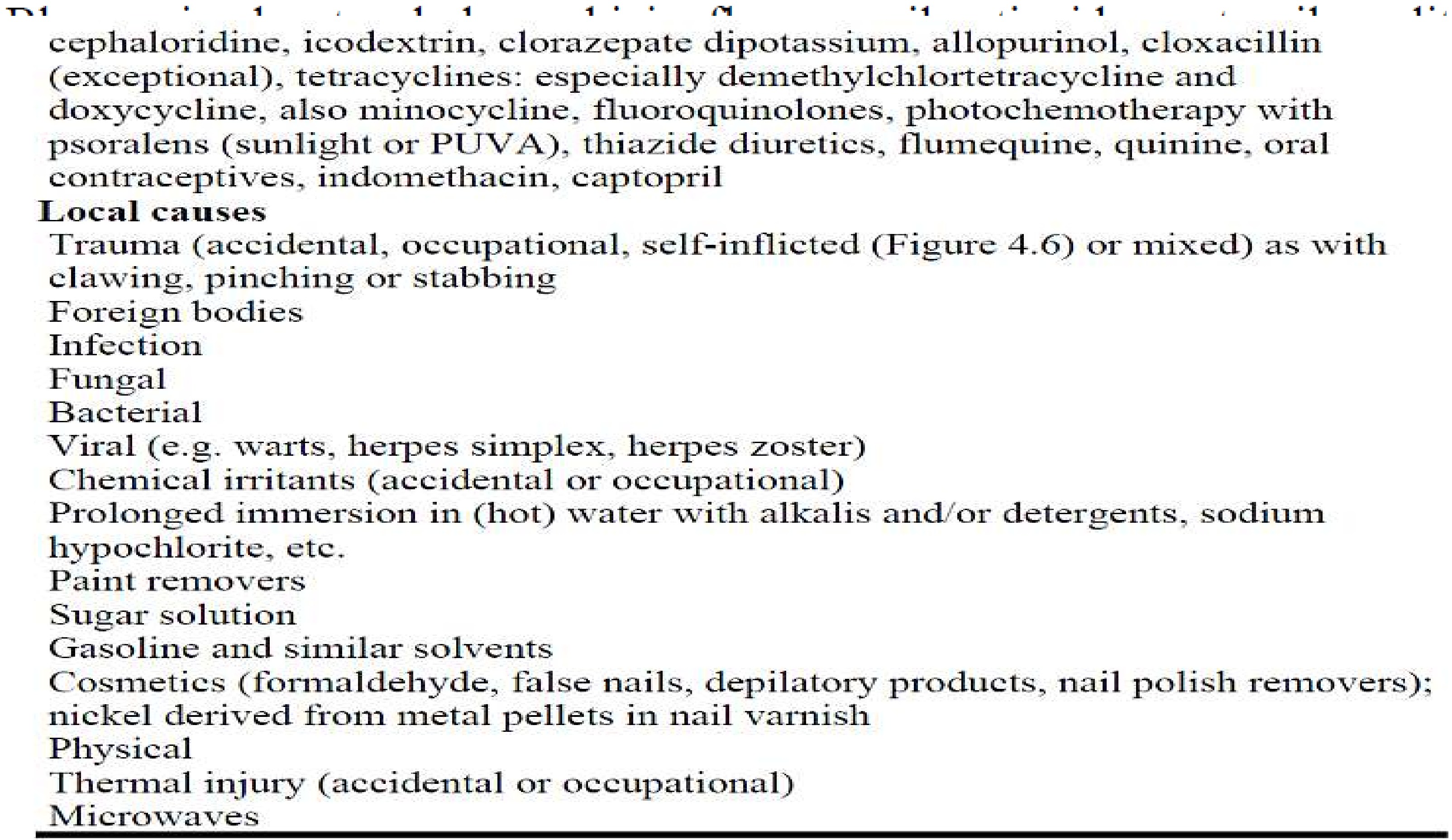
Hyperhidrosis—tumours of the nail bed

Drugs

Bleomycin, docetaxel, doxorubicin, fluorouracil, retinoids, captopril, paclitaxel, mitozantrone

Drug-induced photo-onycholysis: trypaflavin, chlorpromazine, chloramphenicol,

Drugs



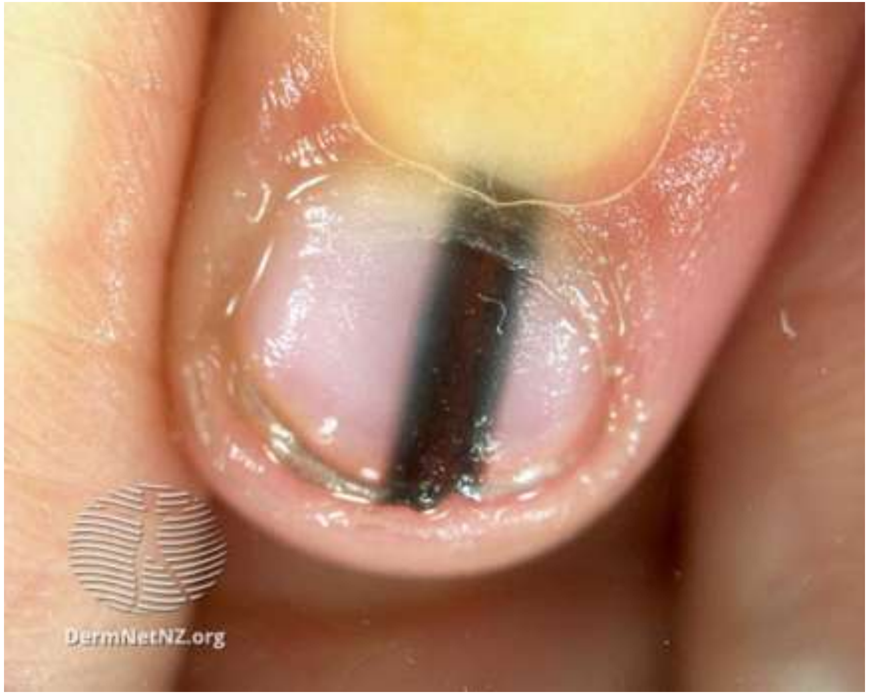
cephaloridine, icodextrin, clorazepate dipotassium, allopurinol, cloxacillin (exceptional), tetracyclines: especially demethylchlortetracycline and doxycycline, also minocycline, fluoroquinolones, photochemotherapy with psoralens (sunlight or PUVA), thiazide diuretics, flumequine, quinine, oral contraceptives, indomethacin, captopril

Local causes

Trauma (accidental, occupational, self-inflicted (Figure 4.6) or mixed) as with clawing, pinching or stabbing
Foreign bodies
Infection
Fungal
Bacterial
Viral (e.g. warts, herpes simplex, herpes zoster)
Chemical irritants (accidental or occupational)
Prolonged immersion in (hot) water with alkalis and/or detergents, sodium hypochlorite, etc.
Paint removers
Sugar solution
Gasoline and similar solvents
Cosmetics (formaldehyde, false nails, depilatory products, nail polish removers); nickel derived from metal pellets in nail varnish
Physical
Thermal injury (accidental or occupational)
Microwaves

Nail Signs Due to Deposition of Pigment

Deposition of Pigment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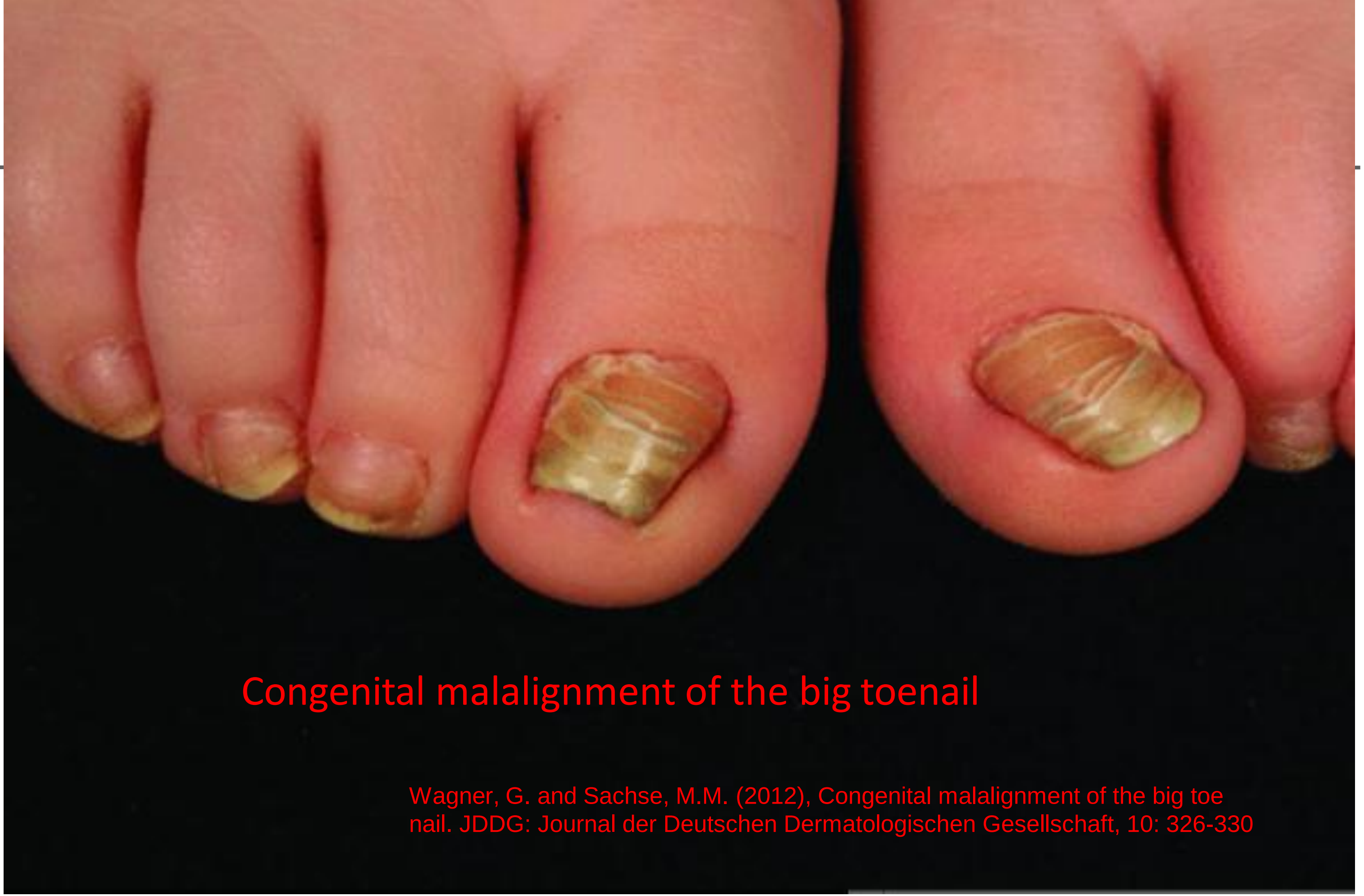
Longitudinal brown to black band

Common in darkly pigmented individuals

Single band may be a sign of melanocyte activation or proliferation (nevus, melanoma)

Multiple bands point to physiologic, trauma/ onychotillomania, drugs or systemic disease

CONGENITAL AND HEREDITARY NAIL DISEASES



Congenital malalignment of the big toenail

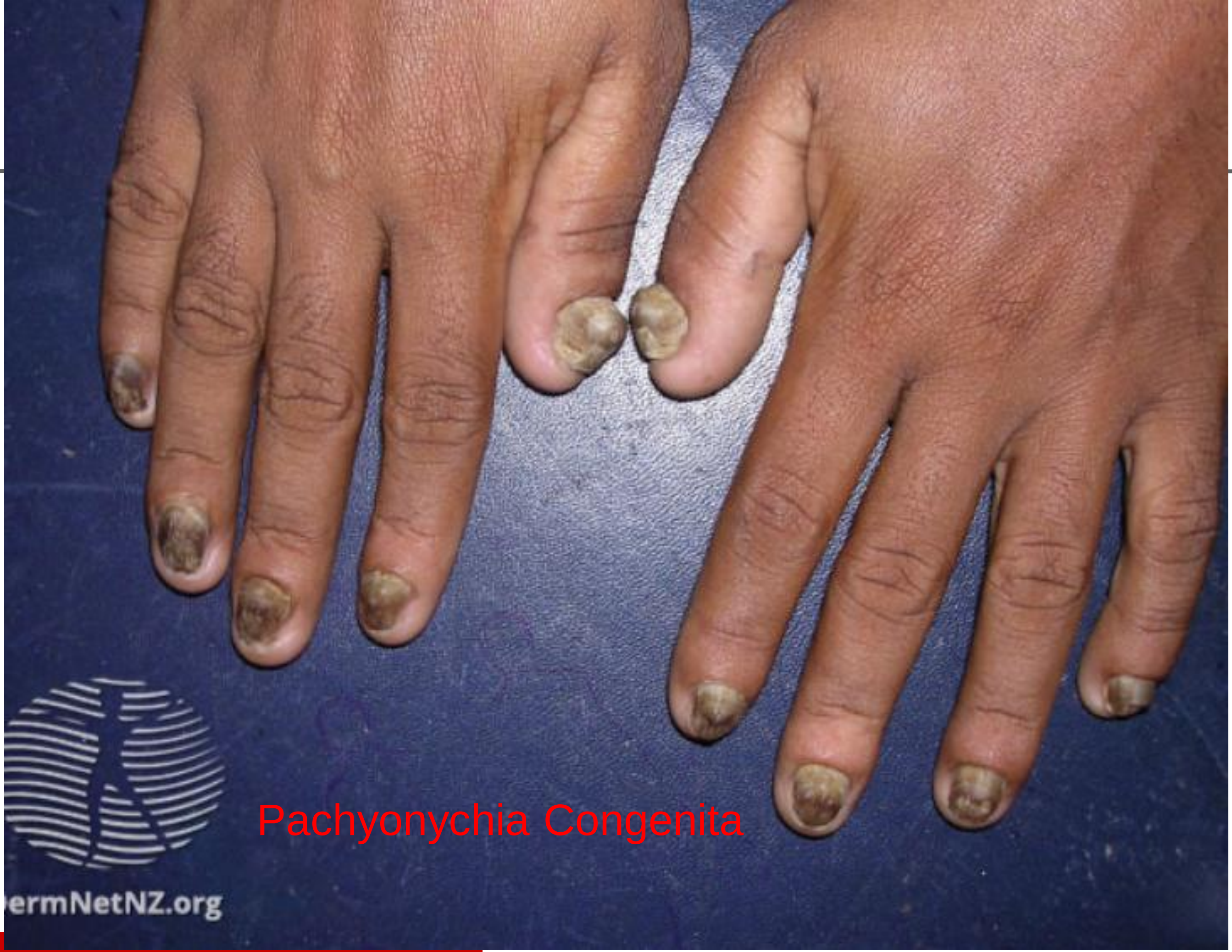
Wagner, G. and Sachse, M.M. (2012), Congenital malalignment of the big toe nail. JDDG: Journal der Deutschen Dermatologischen Gesellschaft, 10: 326-330

Congenital malalignment of the big toenail

Usually bilateral

nail plate of the big toenail deviates laterally from longitudinal axis of the distal phalanx

the nail has a triangular shape, is thick opaque and yellow-brown wih multiple Beau’s lines lateral or distal nail embedding is a common complication noticed by the parents only when the nails are severely dystrophic or ingrown


Pachyonychia Congenita

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Pachyonychia Congenita

The characteristic finding is a thickened nail with an increased transverse curvature due to severe nail bed hyperkeratosis. Affected nails are extremely hard and difficult to trim.

THE NAIL IN DERMATOLOGIC DISEASES

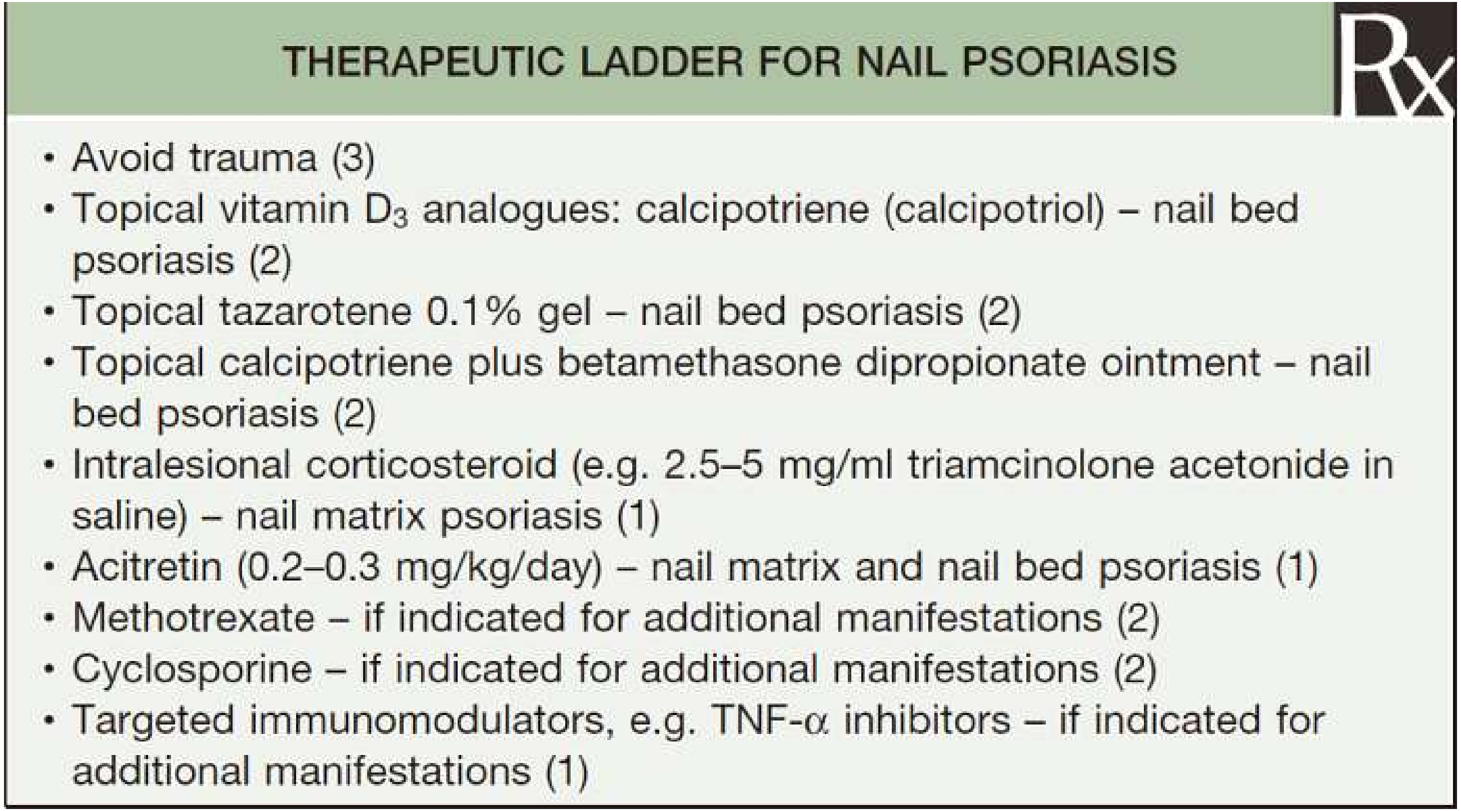
Psoriasis

irregular pitting, nail bed“oil drops”, and

onycholysis with an erythematous border

(diagnostic) other abnormalities include
splinter hemorrhages, subungual hyperkeratosis,
nail plate thickening and crumbling, and paronychia

Treatment



THERAPEUTIC LADDER FOR NAIL PSORIASIS

- Avoid trauma (3)
- Topical vitamin D_3 analogues: calcipotriene (calcipotriol) – nail bed psoriasis (2)
- Topical tazarotene 0.1% gel – nail bed psoriasis (2)
- Topical calcipotriene plus betamethasone dipropionate ointment – nail bed psoriasis (2)
- Intralesional corticosteroid (e.g. 2.5–5 mg/ml triamcinolone acetonide in saline) – nail matrix psoriasis (1)
- Acitretin (0.2–0.3 mg/kg/day) – nail matrix and nail bed psoriasis (1)
- Methotrexate – if indicated for additional manifestations (2)
- Cyclosporine – if indicated for additional manifestations (2)
- Targeted immunomodulators, e.g. TNF-α inhibitors – if indicated for additional manifestations (1)

Lichen planus

, nail thinning, longitudinal ridging and fissuring, and dorsal pterygium (due to scarring). The combination of fissuring plus atrophy can lead to a deformity likened to “angel’s wings”.

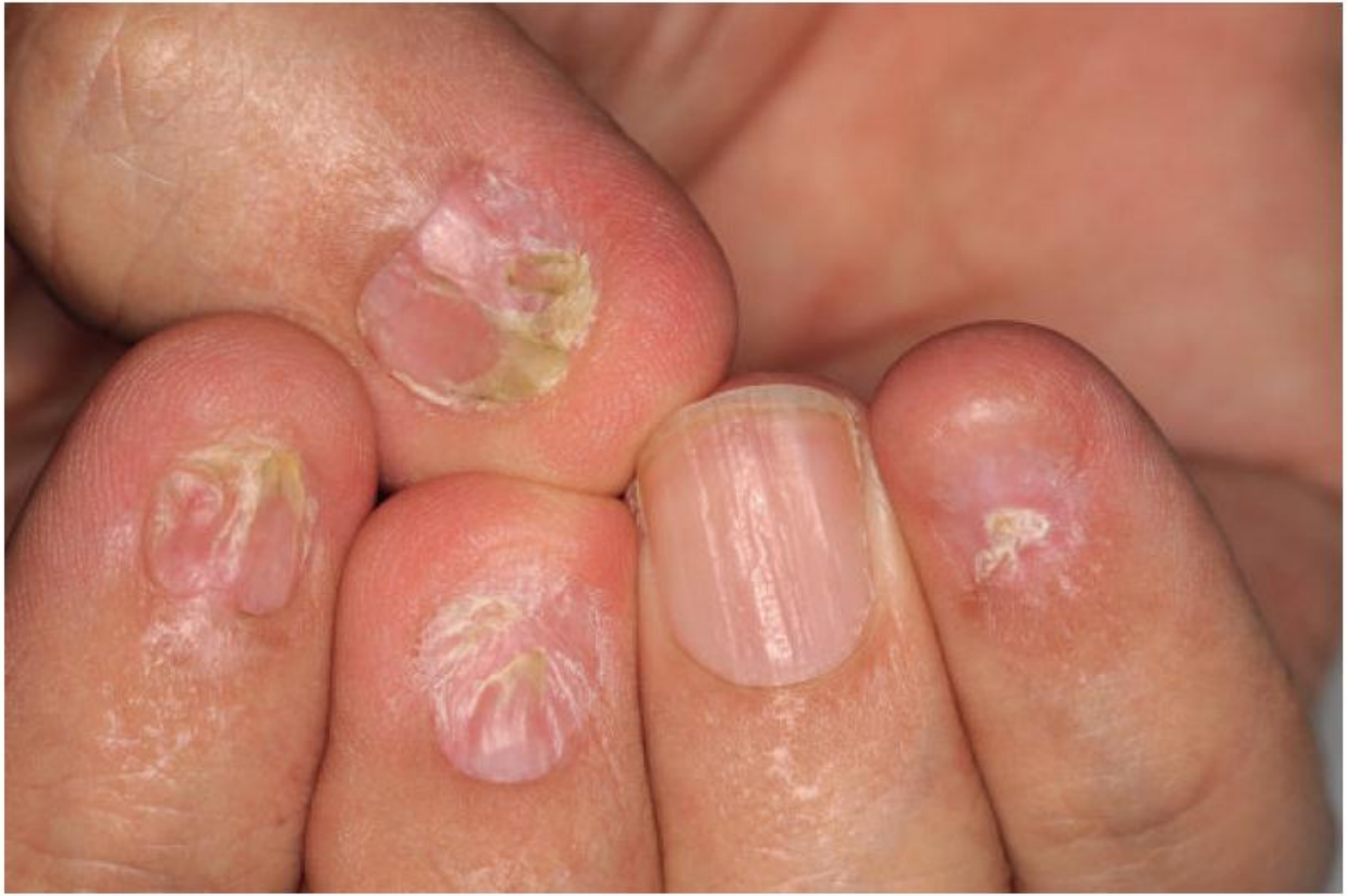


Fig. 71.15 Nail lichen planus. Note the nail thinning, atrophy, and dorsal pterygium. The index finger has an “angel’s wings” deformity.

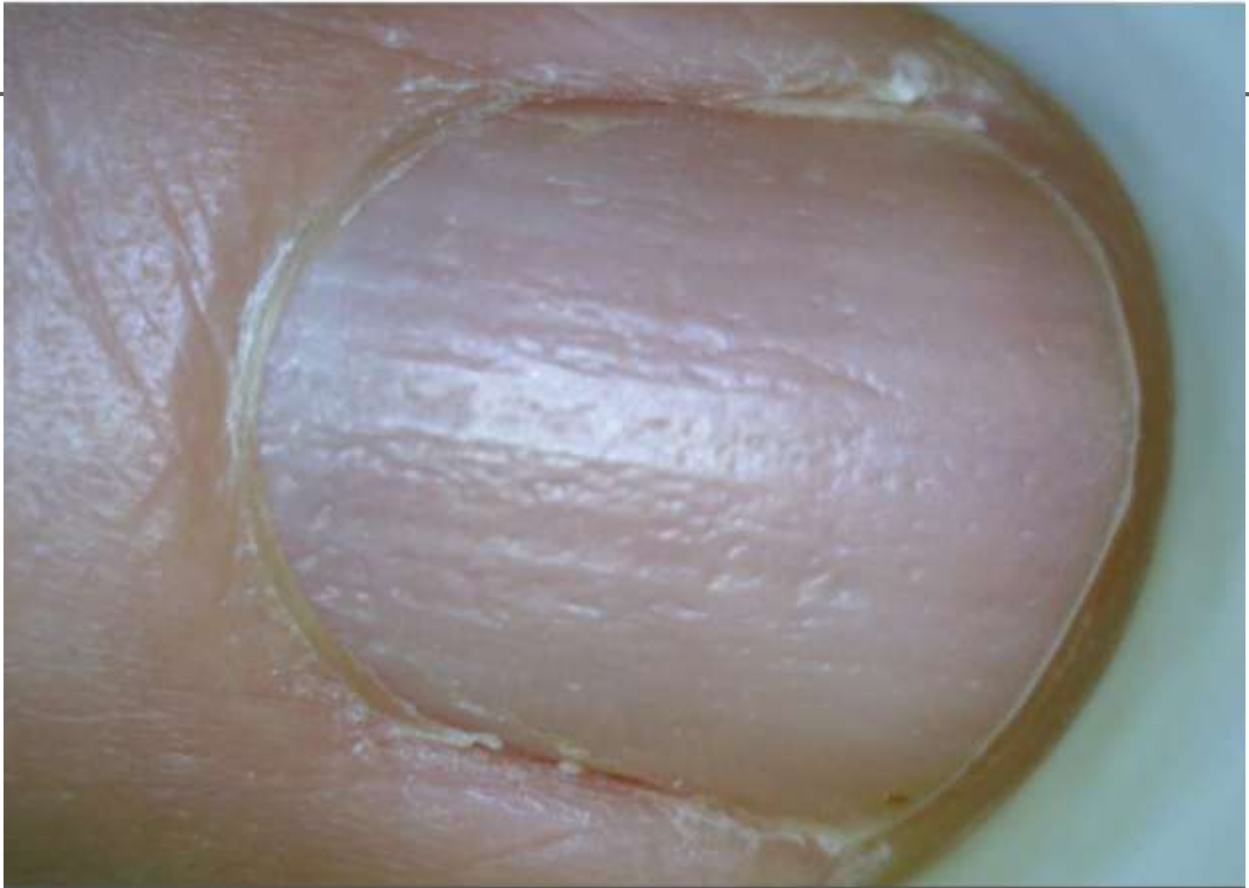
Treatment

Treatment with systemic corticosteroids may be required to avoid pterygium formation. Intralesional injections of corticosteroid (2.5–5 mg/ml triamcinolone acetonide in saline) may be utilized when the disease involves a few nails.

Alopecia areata

geometric pitting, trachyonychia, punctate leukonychia, erythema of the lunula, and onychomadesis

Treatment : cluding oral, topical and intralesional corticosteroids, immunosuppressants, cyclosporin, PUVA, and Jak inhibitor



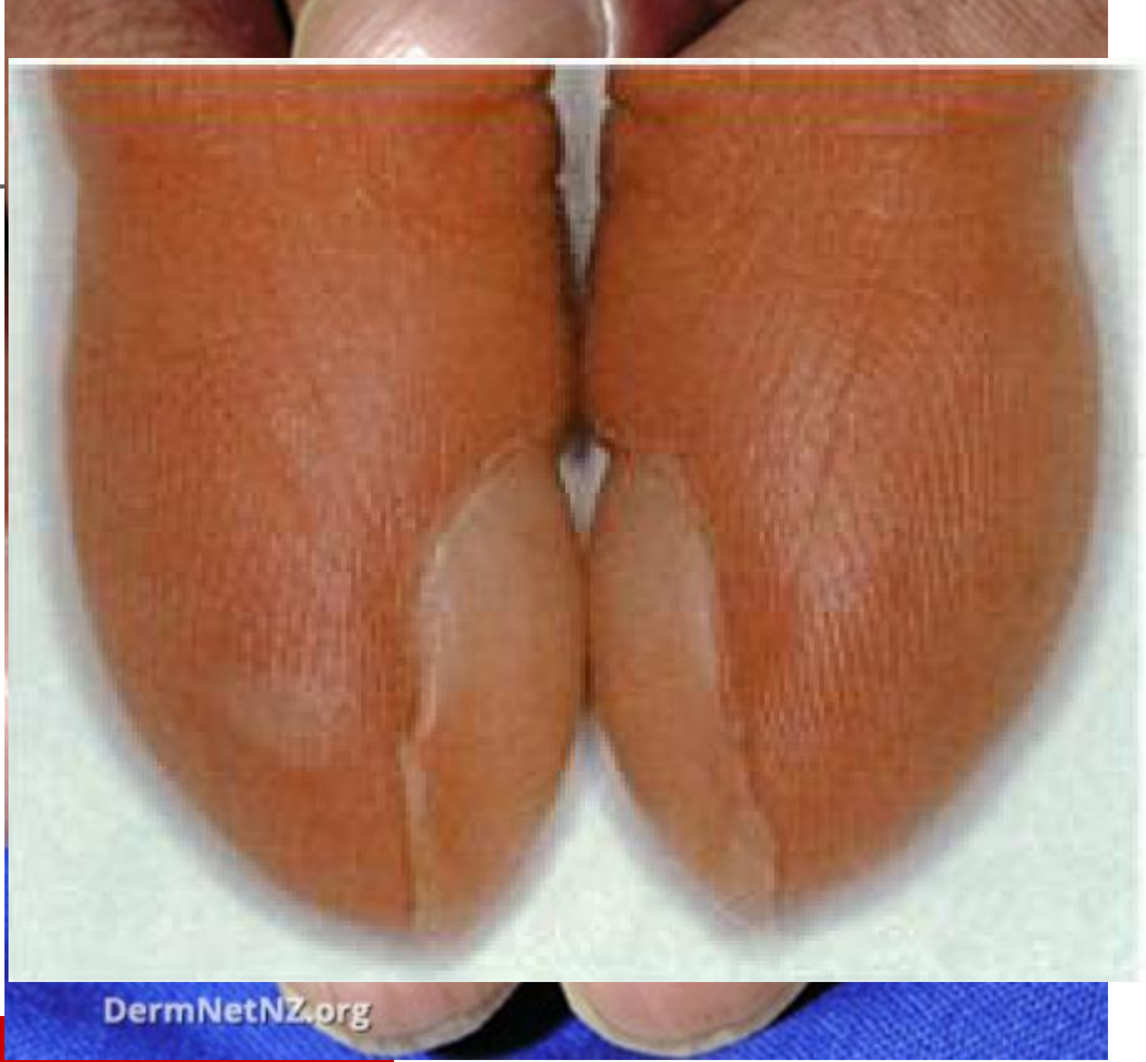
Nail changes, particularly nail pitting, are common in patients with alopecia areata

Eczema

pitting and Beau’s lines; onychomadesis, subungual hyperkeratosis,onycholysis, and fissuring of the hyponychium

Controlling the skin disease results in gradual improvement of the nails.

THE NAIL IN SYSTEMIC DISEASES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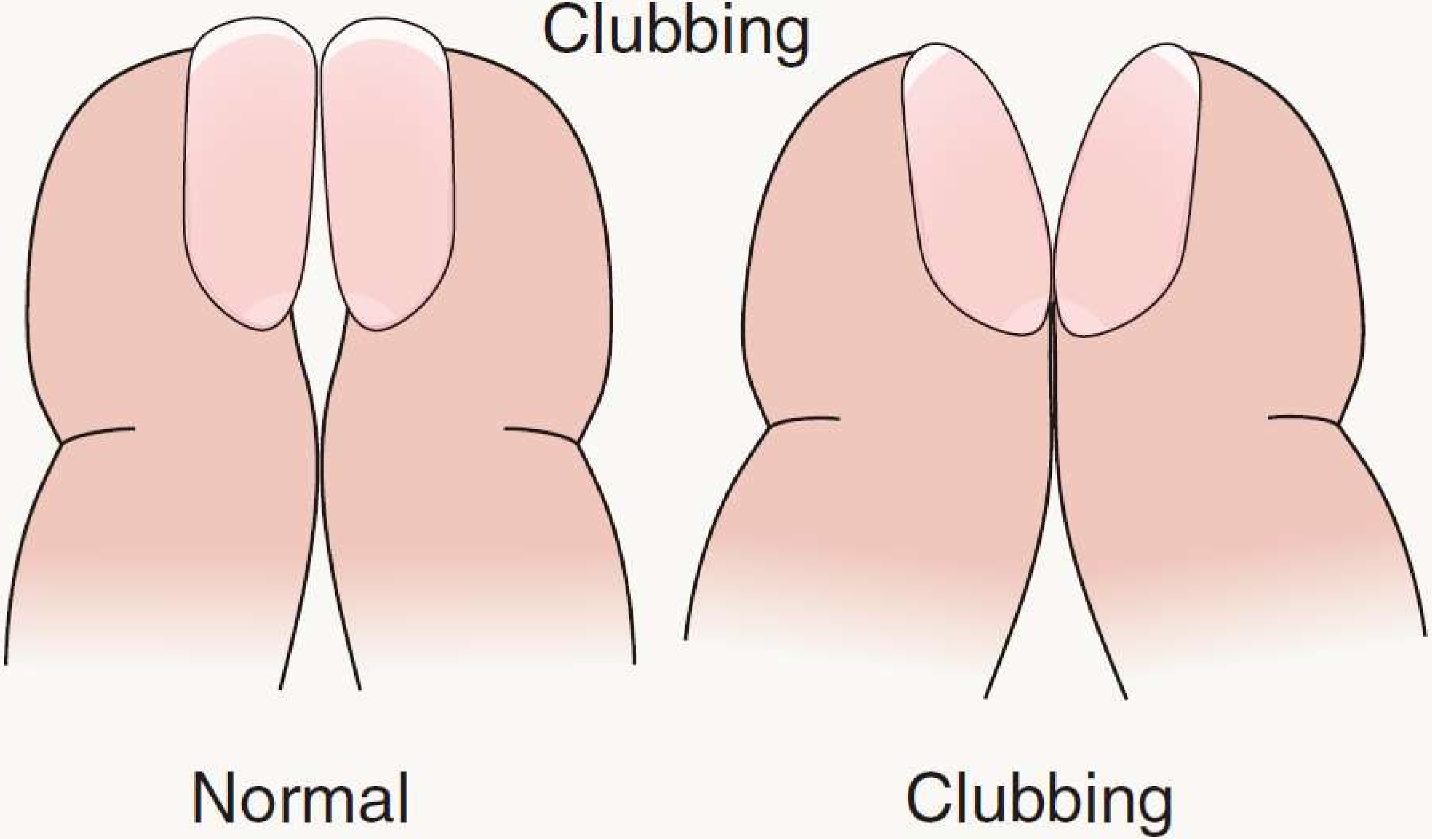
Clubbing


Normal
Clubbing

1.Clubbing (Watch-Glass Nails, Hippocratic Fingers, Drumstick Fingers)

Clubbing is caused by enlargement of the soft tissue of the distal digit, leading to a bulbous appearance. The nail plate is enlarged and
excessively curved, with widening of the angle between the proximal
nail fold and the nail plate to greater than 180° (Lovibond’s sign)

Table 2.1 General causes of clubbing and pseudoclubbing

Clubbing	*Pseudoclubbing*
Unilateral	Yellow nail syndrome (Figure 2.7)
Aortic/subclavian aneurysm	Gout
Brachial plexus injury	Sarcoidosis
Trauma (Figure 2.5)	Osteoid osteoma
	Perineurioma
	Metastases
Lower extremities	Congenital abnormalities
Arterial graft sepsis	Chronic paronychia—severe hook nail
General	
Congenital	
familial/sporadic	
Thoracic tumours (bronchopulmonary cancers)	
Pulmonary	
Cardiovascular	
Gastrointestinal	
inflammatory bowel disease	
parasitosis	
liver disease	
tropical sprue	
Endocrine/metabolic	
thyroid, acromegaly (Figure 2.6)	
malnutrition	
AIDS	
Secondary to pulmonary and other infections	

2. Yellow Nail Syndrome

Yellow nail syndrome is an uncommon disorder of unknown etiology

Linear nail growth is arrested or greatly reduced. The nails are thickened and transversely and longitudinally overcurved with disappearance of the cuticle. The nail color varies from pale yellow to dark yellow–green

Characteristically, the nail abnormalities are associated with lymphedema

and respiratory tract involvement, including chronic bronchitis, bronchiectasis, sinusitis, and pleural effusions.

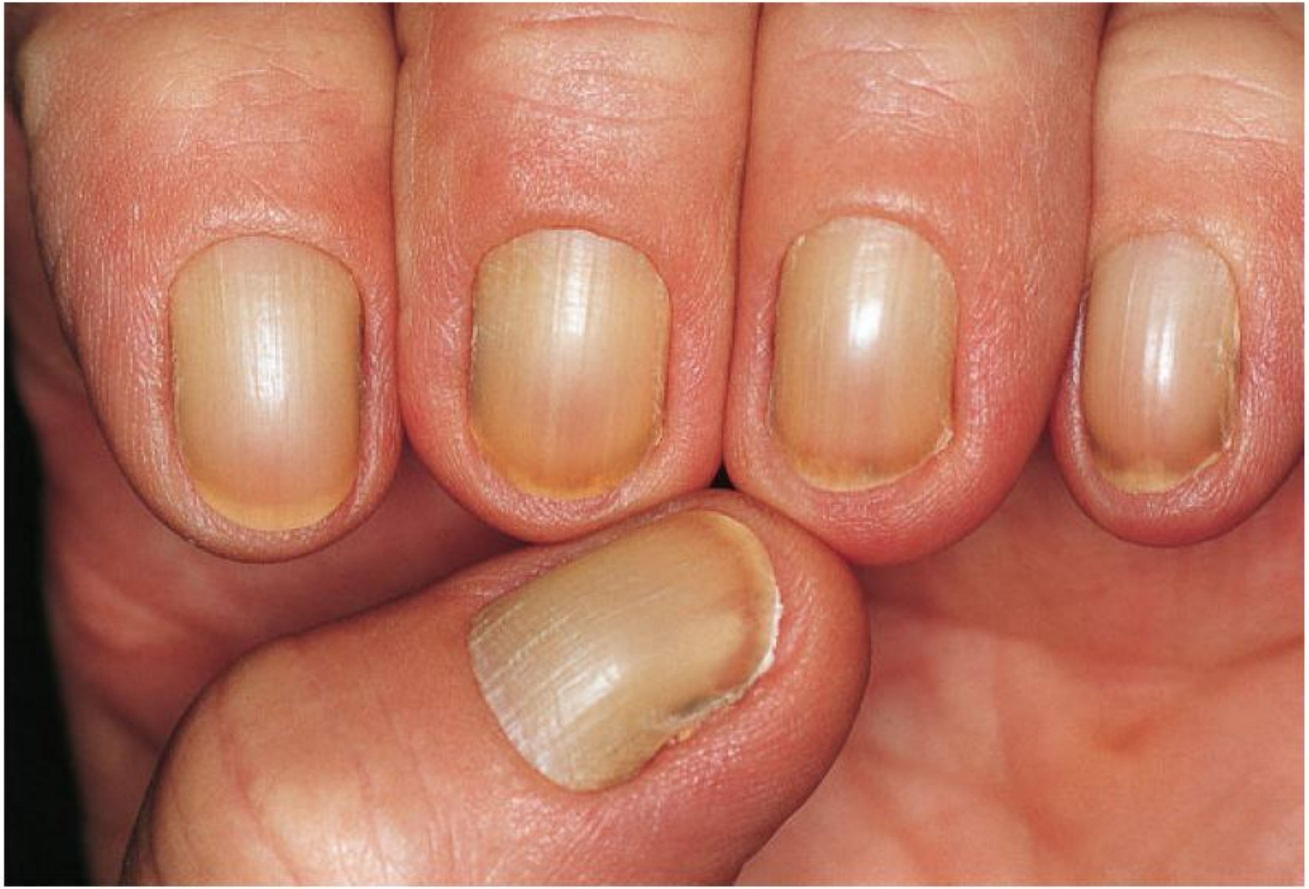


Fig. 71.16 Yellow nail syndrome. Note the overcurvature of the nail, both transversely and longitudinally, and loss of the cuticle, in addition to the yellow discoloration.

Treatment

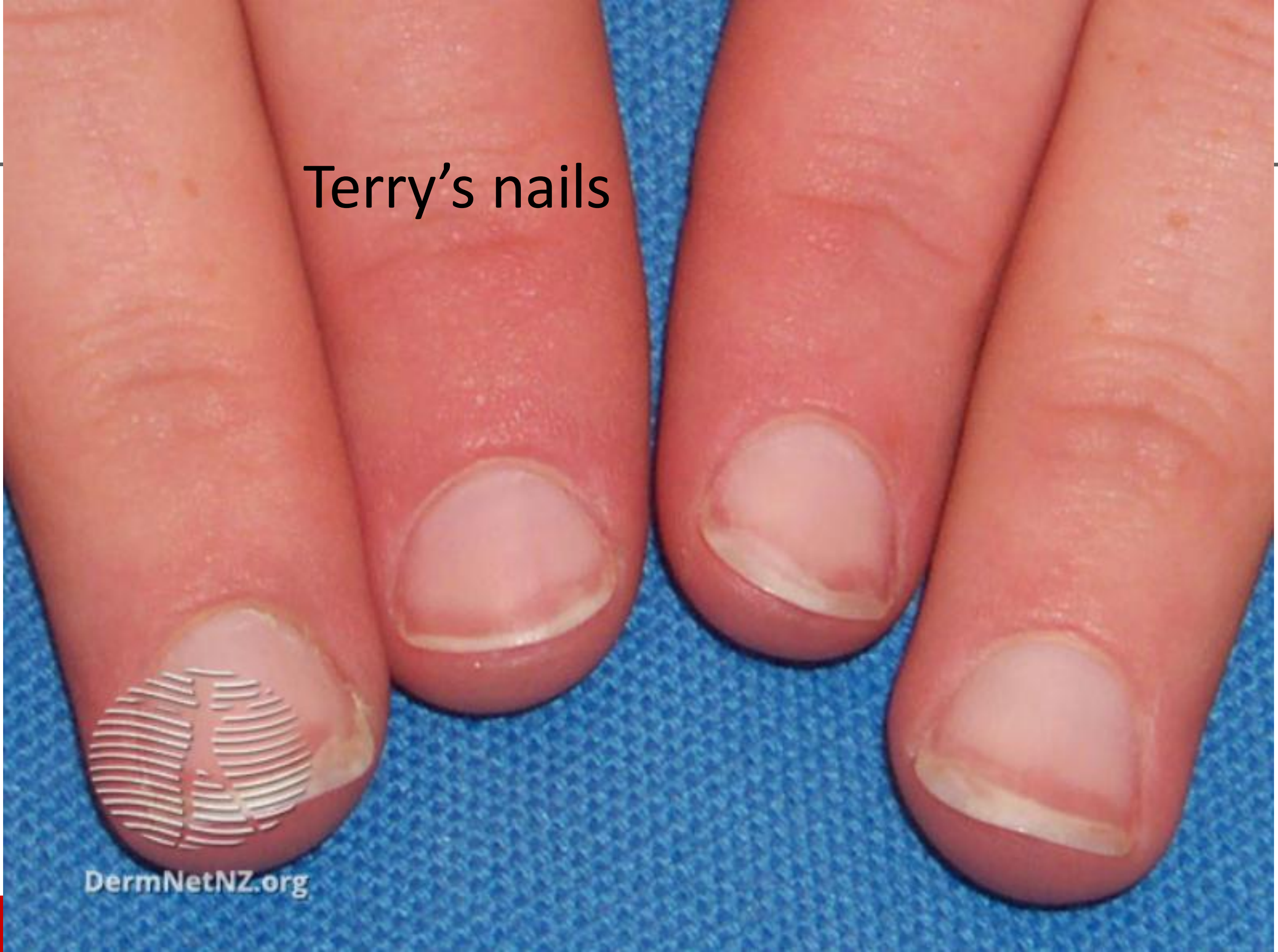
Treatment is not effective in all cases and must be prescribed for several
months19: • vitamin E 1200 IU/day • pulse itraconazole (200 mg twice daily for 1 week a month) or
fluconazole (150 mg daily for 1 week a month)

Muehrcke's nails: chemotherapy

Half-and-half nails : chronic renal disease

Terry's nails: liver cirrhosis

Terry's nails



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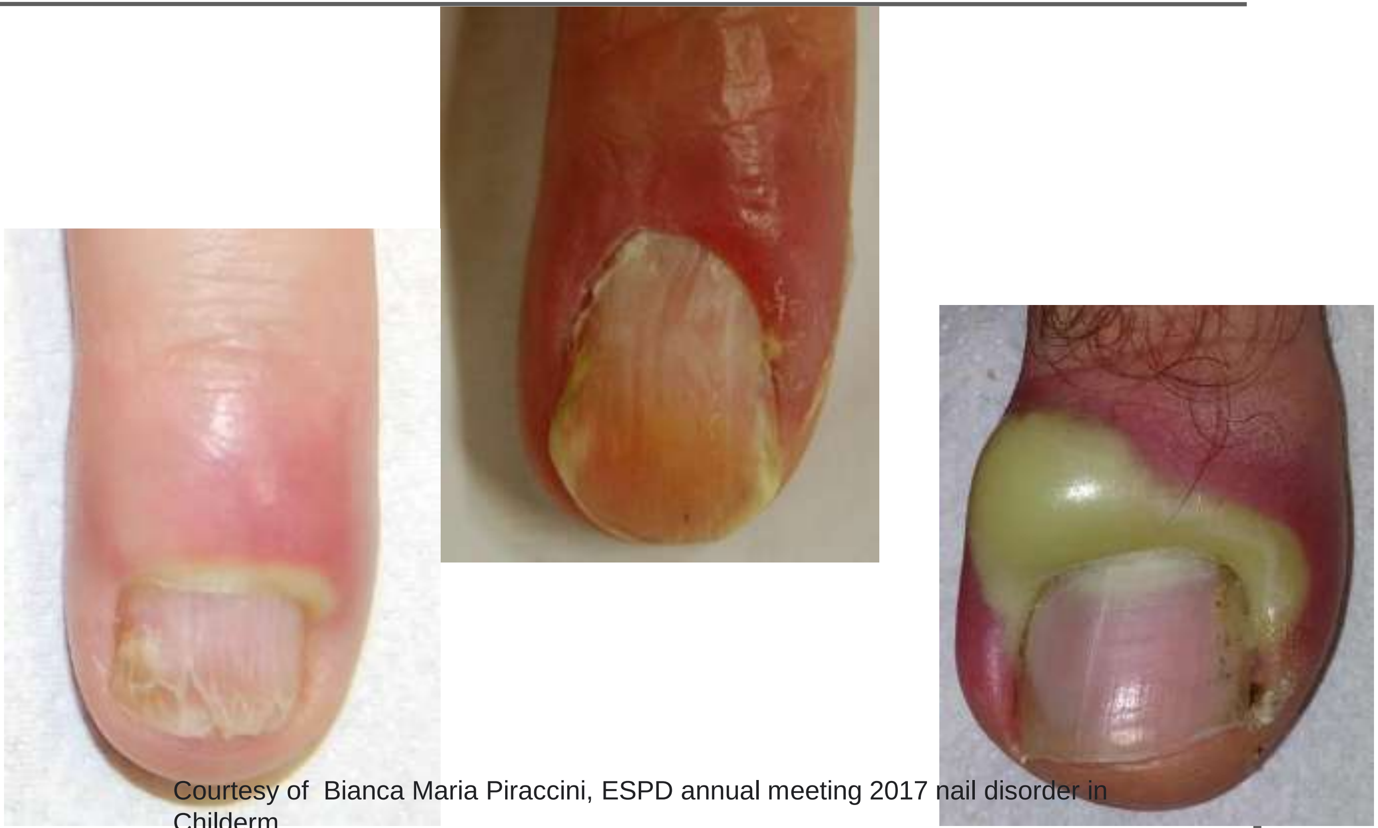


Courtesy of Bianca Maria Piraccini, ESPD annual meeting 2017 nail disorder in Childerm

Acute paronychia

The affected digit becomes swollen, red, and painful purulent drainage.

Acute paronychia is most commonly due to bacteria, in particular Staphylococcus aureus or Streptococcus pyogenes

follows minor trauma to the nail

Recurrent episodes of acute paronychia should raise the suspicion of an HSV

Treatment

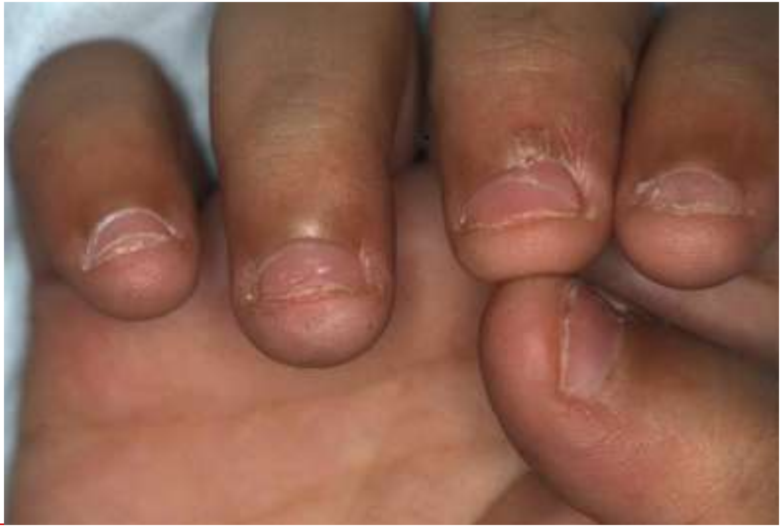
Drainage of the abscess, followed by systemic antibiotics (based upon culture results) or systemic antivirals when due to HSV.

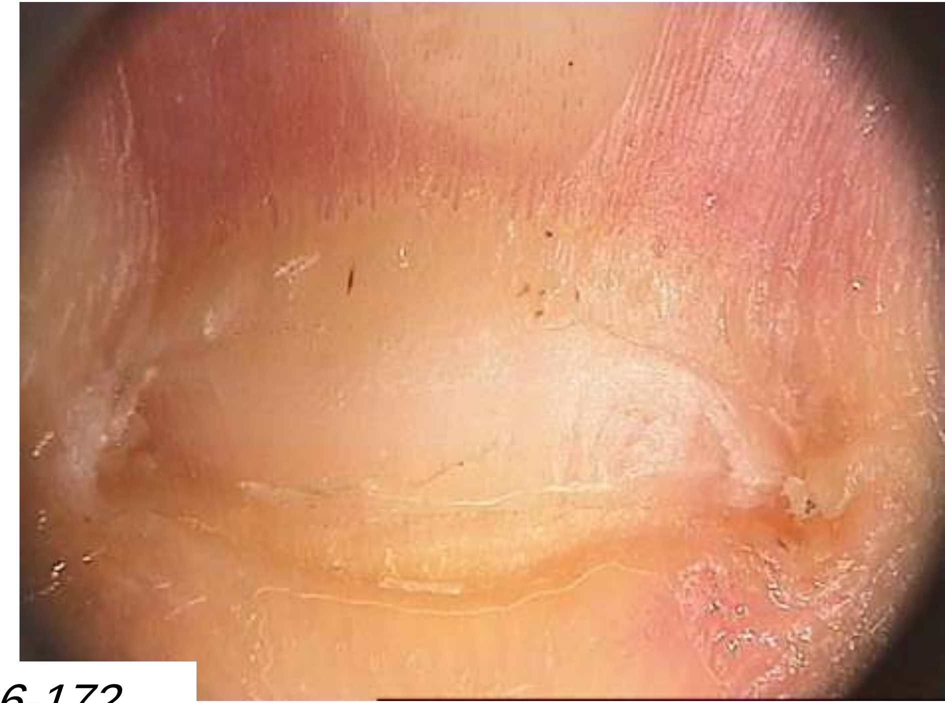
Nail biting

Rare before the of 3 years, peaks in childhood and adolescence

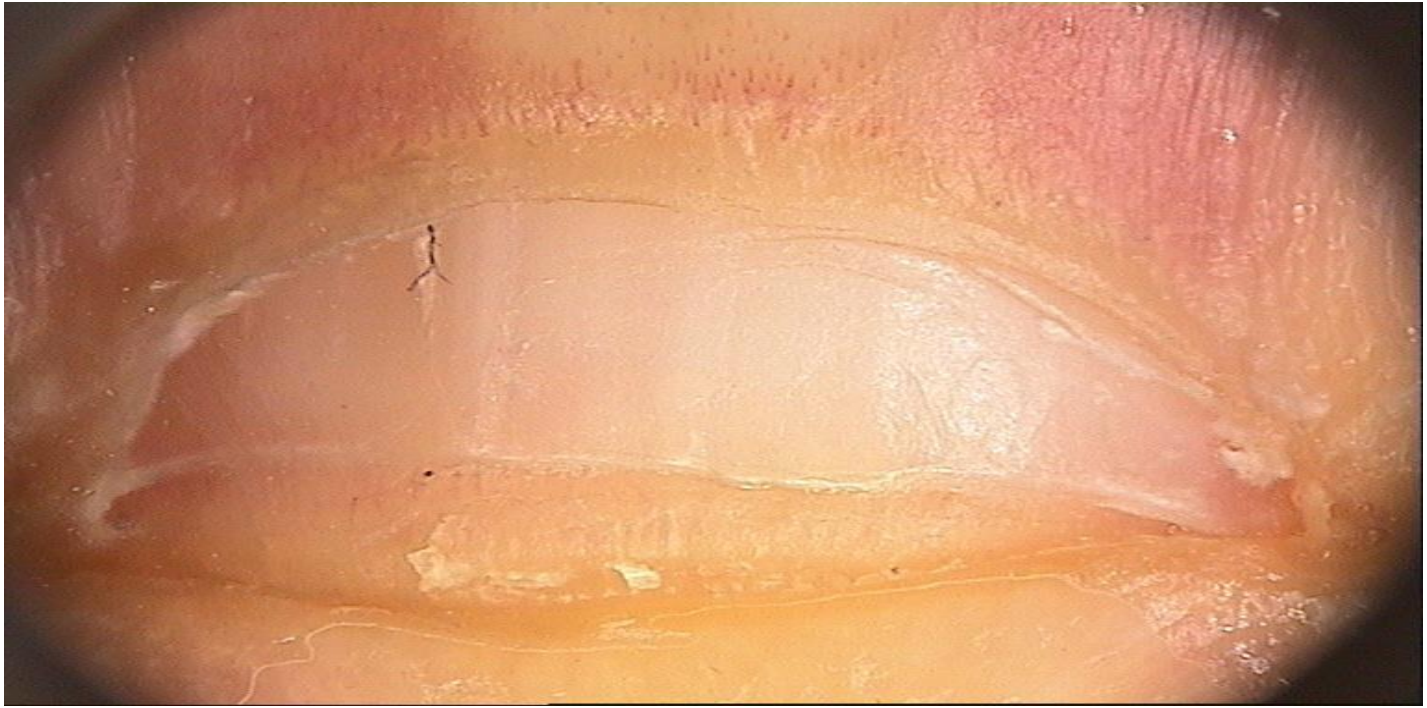
prevalence of about 30% at around 6 year of age

occurs as a result of boredom or working on difficult problems rather than anxiety





Halteh et al. J Dermatolog Treat. 2017;28: 166-172.

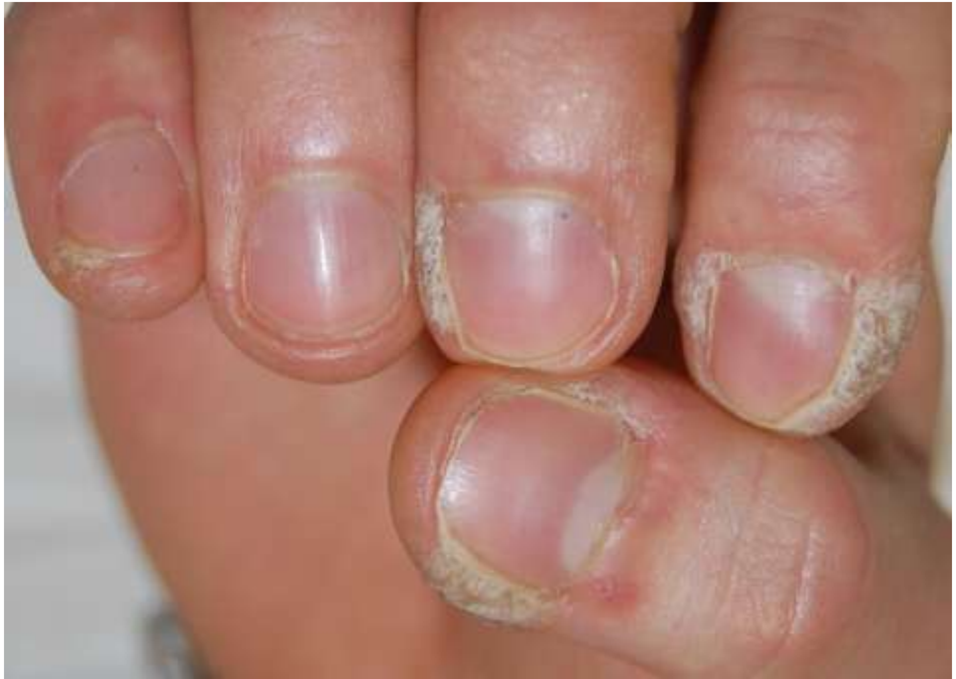


Shortening of fingernail, with transverse axes longer than longitudinal ones, the hyponychium and nail bed skin become visible

Nail biting

Complications

- change in the oral bacteria (< Enterobacteriaceae)
- dental problems: apical root resorption, alveolar destruction, malocclusions, temporo-mandibular disorders and gum injuries.
- negative social and psychological consequences for the patients and their parents



Periungual warts are a common complication!

Nail biting

Treatment

- Non-pharmacological therapies

Treatment method	Definition
Punishment	Reprimanding the nail biting behavior with an aim to stop the activity (60)
Habit reversal	Encompasses awareness of the nail bitingactivity, warning signs, and development of a distracting competing response (62)
Object manipulation	Manipulation of an object during warning signs of nail biting (67)
Bitter tasting lacquer	Aversive therapy used to deter from nail biting due to bad taste of the substance (68)
Non-removable reminder	Wrist-band worn as a non-removable reminder to hinder nail biting (71)

Halteh et al. J Dermatolog Treat. 2017;28: 166-172.

Nail biting

Treatment

Wristbands provided by
entWristbands.com

- bitter tasting lacquers (containing denatonium benzoate and sucrose octaacetate)
- non-removable reminder technique

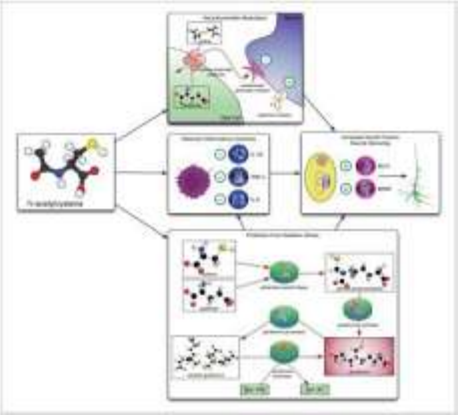
MAVALA
MAVALA STOP
DISCOURAGES NAIL BITING AND THUMB SUCKING
Nail biting ?
10 ml
MAVALA
SWITZERLAND
MAVALA STOP
MAVALA

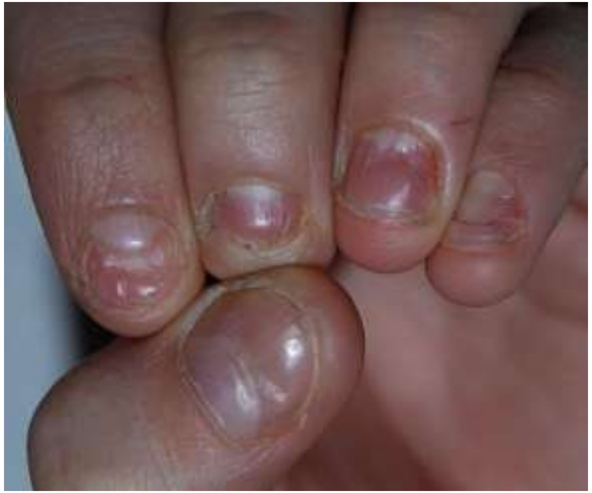
Koritzky & Yechiam. Behav Modif. 2011; 35: .511-30

Nail biting

Treatment

- N-acetyl cysteine (NAC) 800-1200 mg/day





Ghanizadeh et al. Antiinflamm Antiallergy Agents Med Chem. 2013; 12: 223-8.
Deanety al. J Psych Neurosc 2011; 36: 78–.86

Thanks

For more Question feel free to contact me
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DERMATOLOGY LECTURE NOTES FOR MEDICAL SCHOOL

DR. SAHAR AHMED AL-AWDI

DERMATOLOGY DEPARTMENT; FACULTY OF MEDICINE

SANA’A UNIVERSITY

COMMON SKIN INFECTIONS

INFECTIONS OF THE SKIN

- Bacterial infection
- Viral infection
- Fungal infection
- Parasitic infestation
- Mycobacterial infection

FUNGAL SKIN INFECTIONS

Divided into:

1- Superficial fungal skin infection:

a- Dermatophyte infection

b- Candidiasis

c- Malassezia Species (Pityriasis Versicolor)

2- Deep fundal skin infection

FUNGAL SKIN INFECTIONS

1- ***Dermatophytes:***

Dermatophytes are group of fungi capable of infecting nonviable keratins cutaneous structures(st. corneum, nails and hair)

The term tinea is best used for dermatophytes and is modified according to anatomical site.

DERMATOPHYTES:

- Risk factors:

- Hot, humid weather , occlusive footwear; excessive sweating
- Can be transmitted through contact where arthrospores survive for 12 months in human scales.

DERMATOPHYTES:

Diagnosis:

- Potassium hydroxide (KOH): dissolves keratin but leaves behind the hyphae seen under direct microscopy
- Woods lamp
- Fungal culture

DERMATOPHYTES

1- Tinea Capitis (Trichomycosis):

Infection of the scalp

Common fungal infection of children.

- Highly contagious, scalp ringworm most commonly occurs in children, particularly children of African heritage aged 3-9 years.

Scalp involvement: Scaly, black dot , kerion, Favus

Treatment with oral antifungal

TINEA CAPITIS

- Clinical variants:

1- Black dot (Non inflammatory)

2- scaly (Non inflammatory- seborrheic dermatitis like type)

3- kerion (inflammatory)

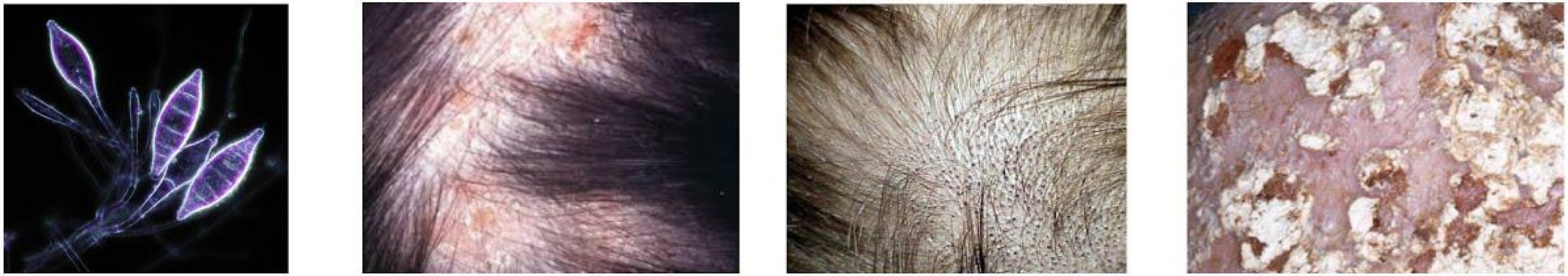
4- Favus (inflammatory)

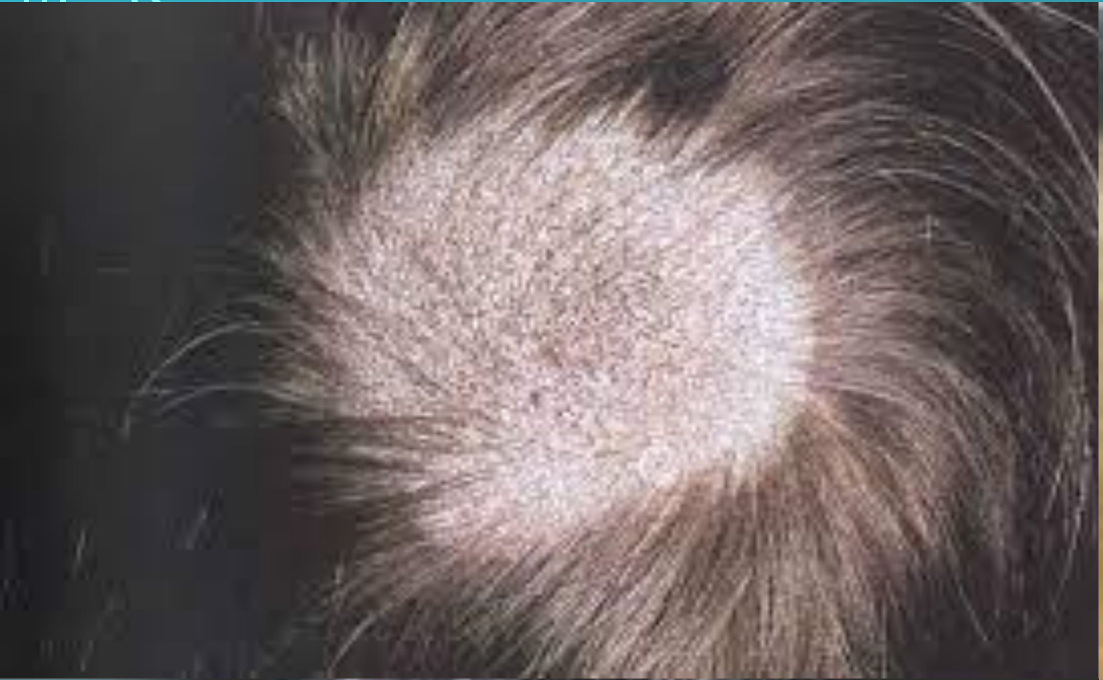
TINEA CAPITIS

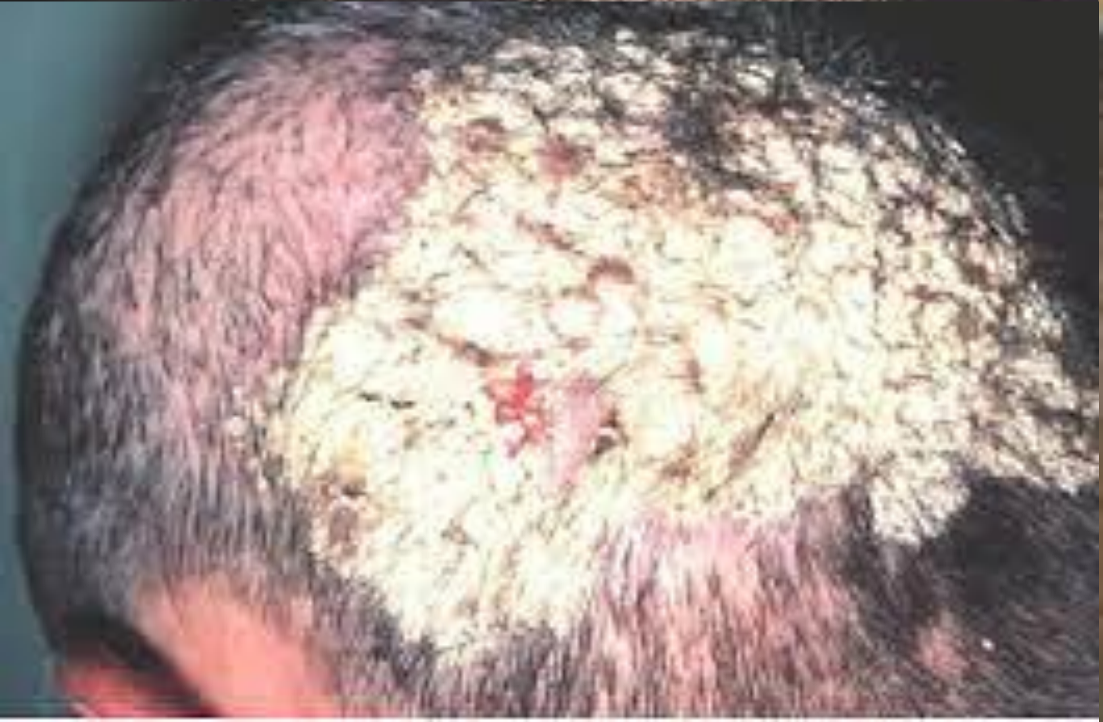
- Spreads through contact with contaminated items and fomits with the spores.
- Managed by topical and systemic ttt

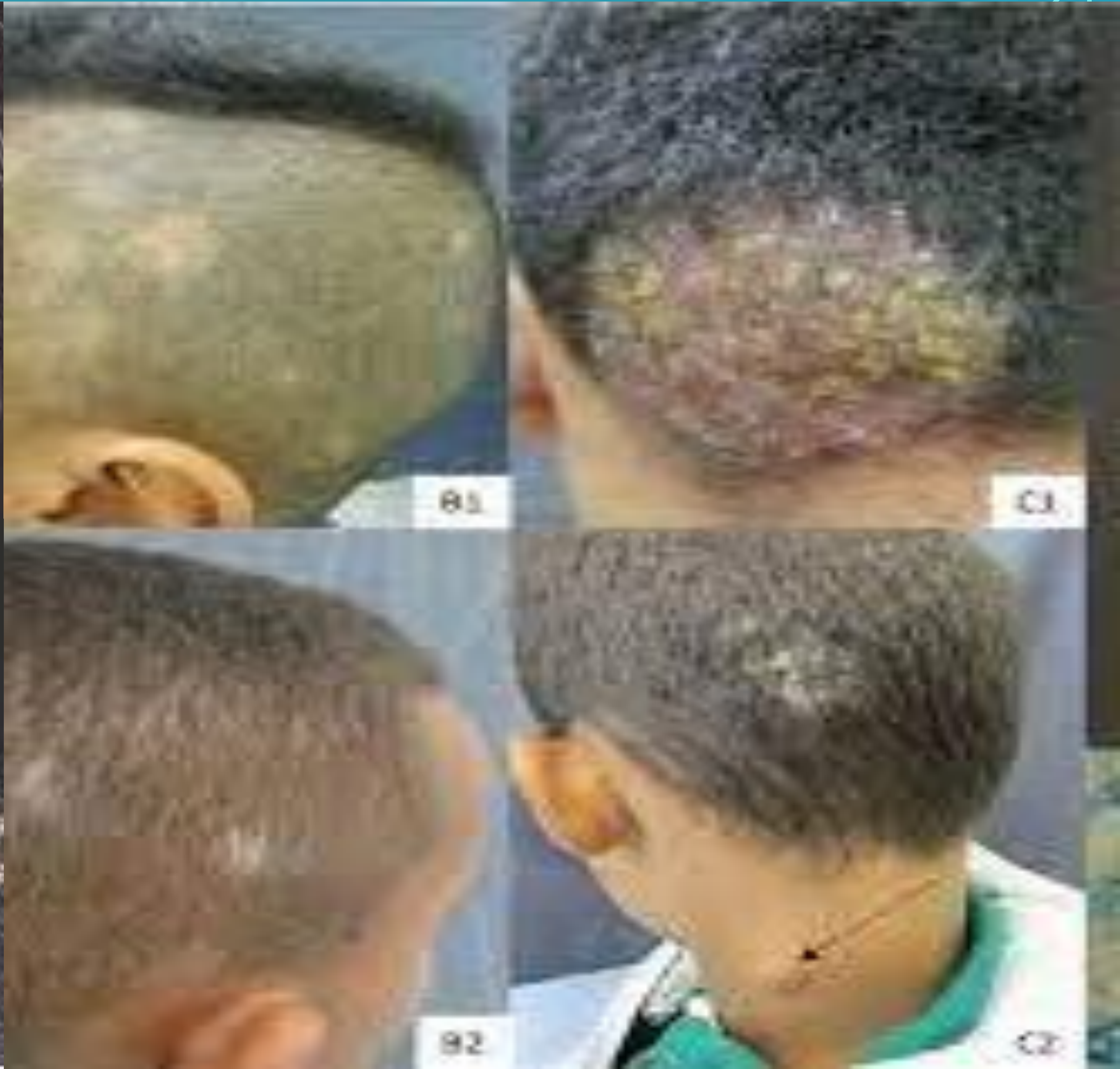
Tinea Capitis

Fungal Infection of Scalp








B1
C1
B2
C2

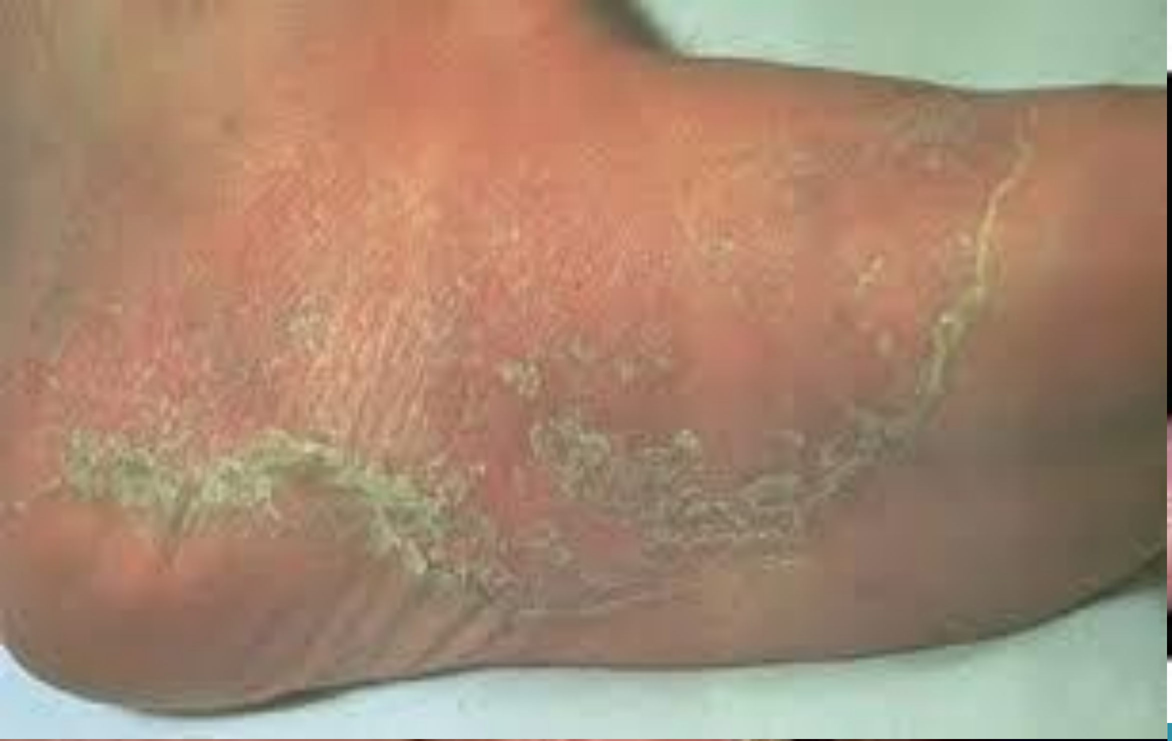
TINEA PEDIS/ATHLETES' FOOT

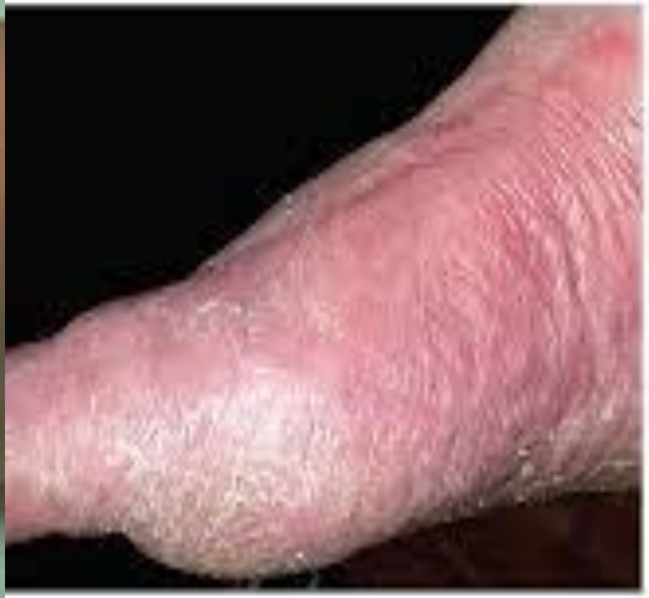
- Dermatophyte infection of the feet
- C/P: erythema, scaling, maceration +/- Bullae
- Clinical variants:-

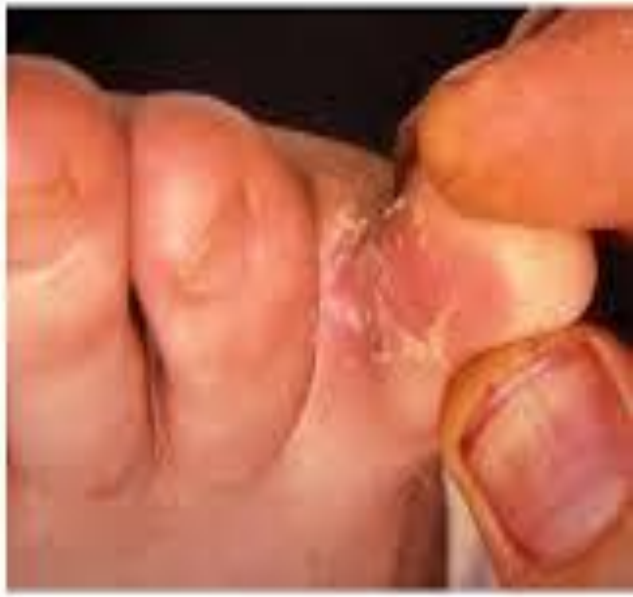
1) Interdigital type (dry, maceration) ;Most commonly b/w fourth and fifth toes.

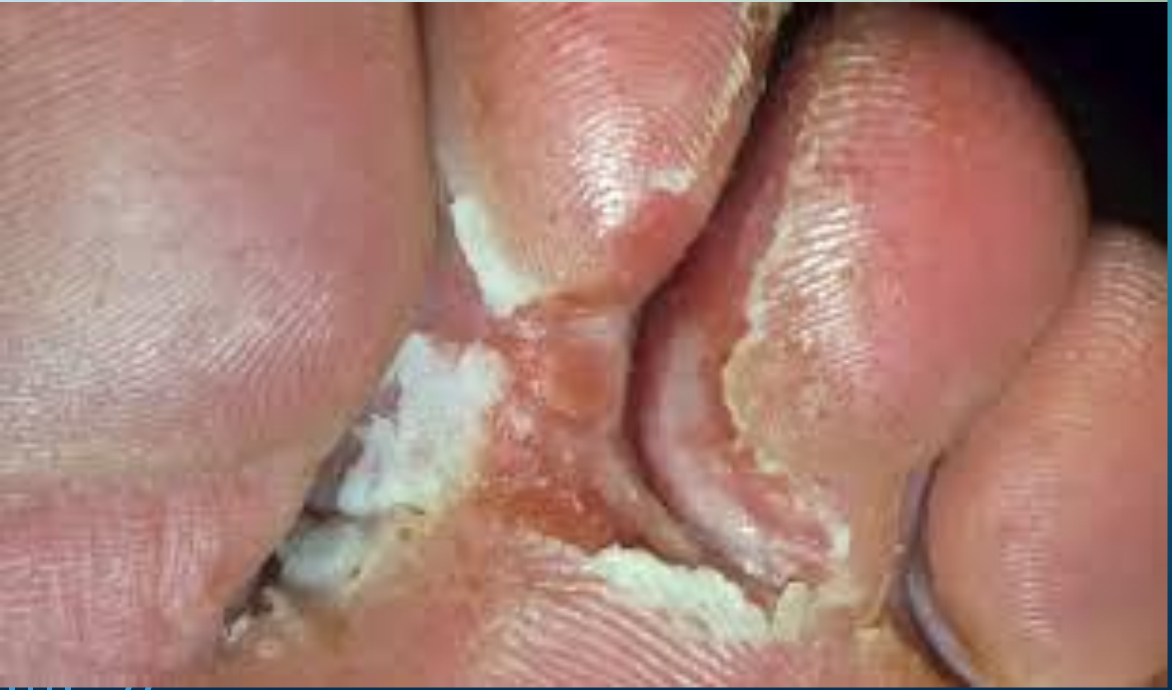
2) Inflammatory bullous type

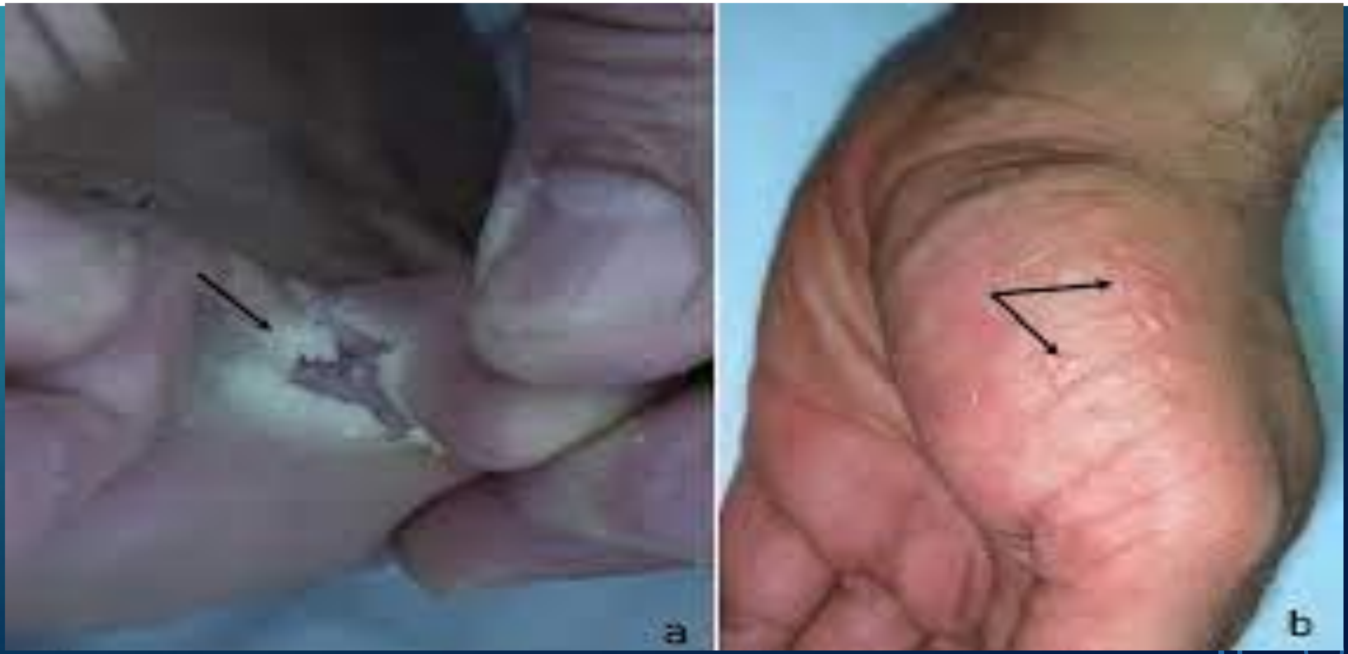
3) Hyperkeratotic type







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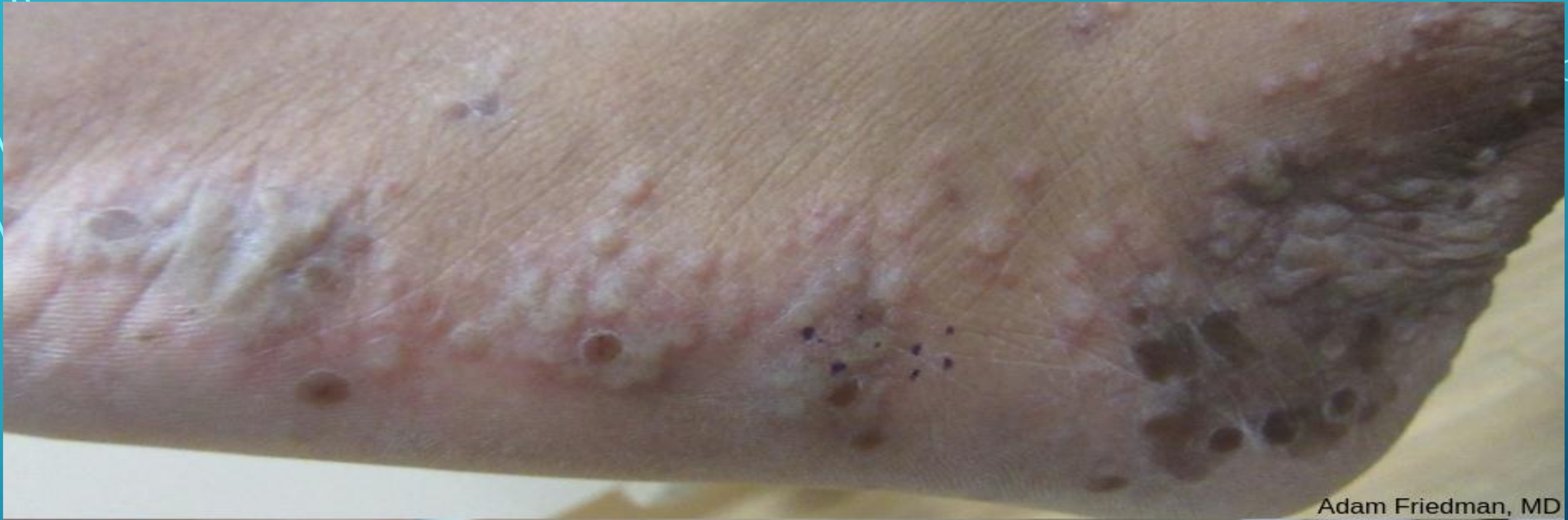
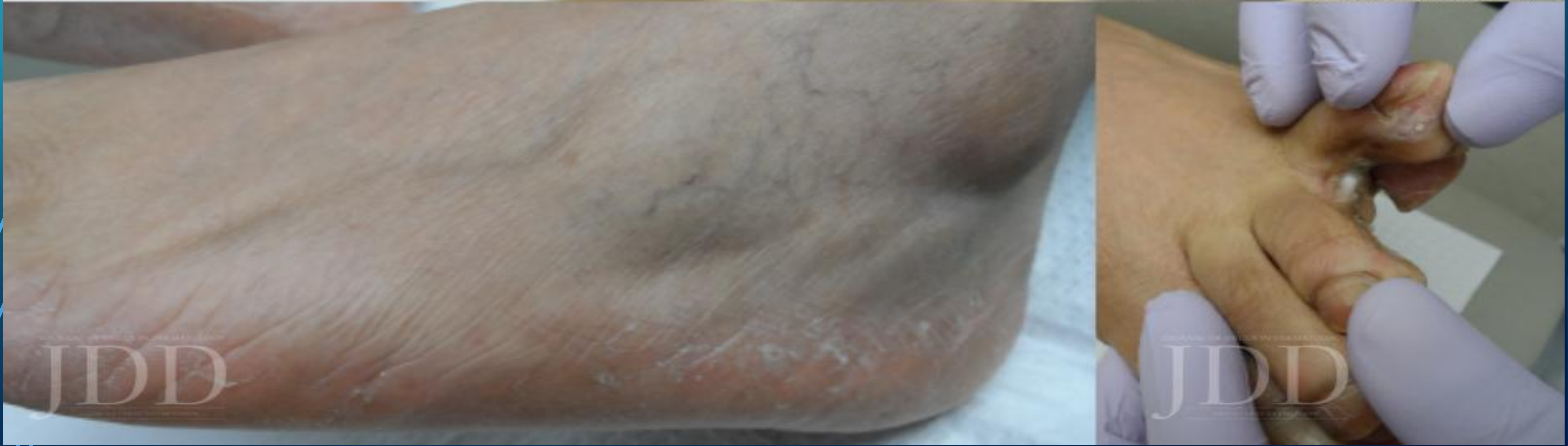


Adam Friedman, MD



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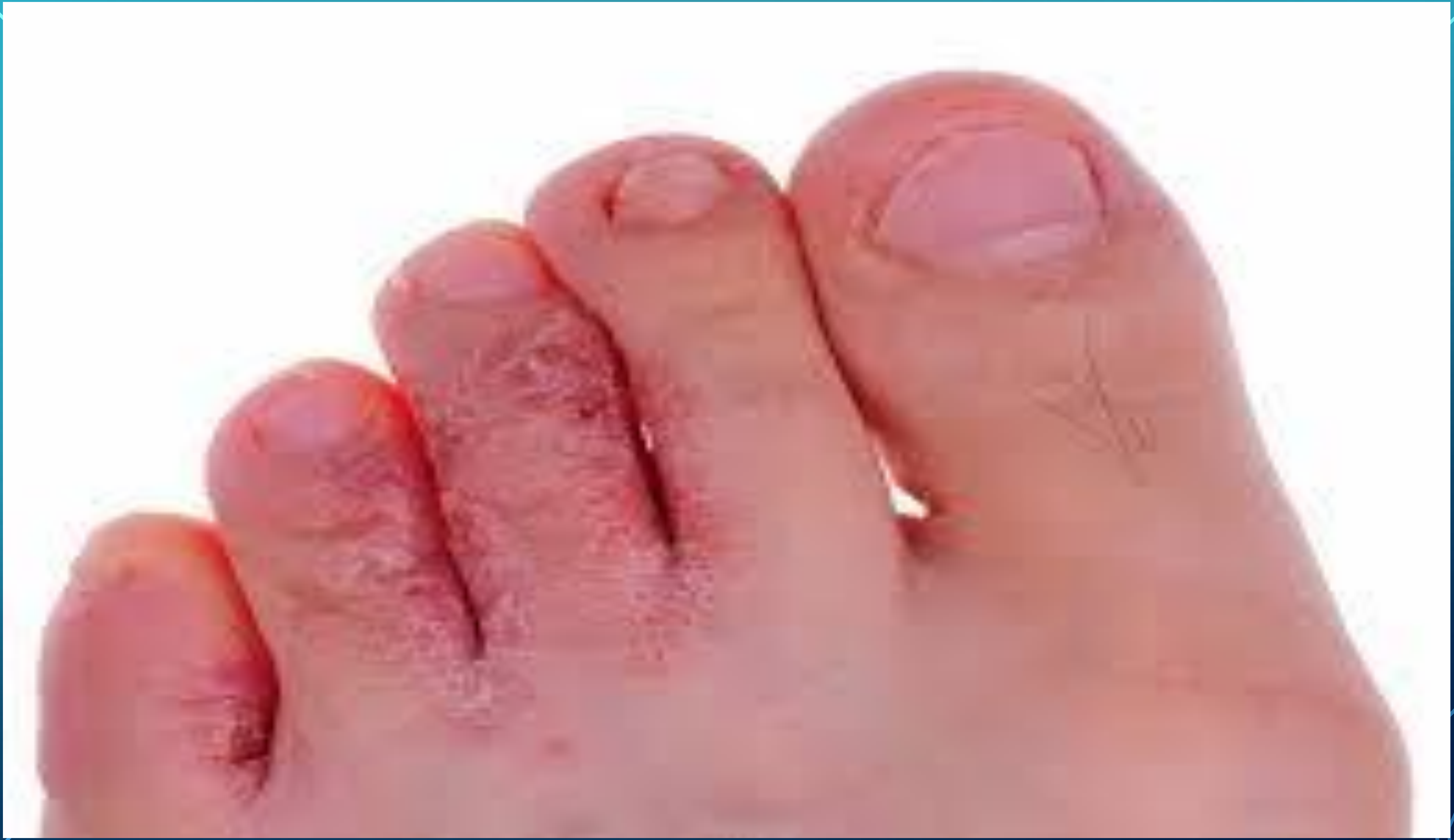
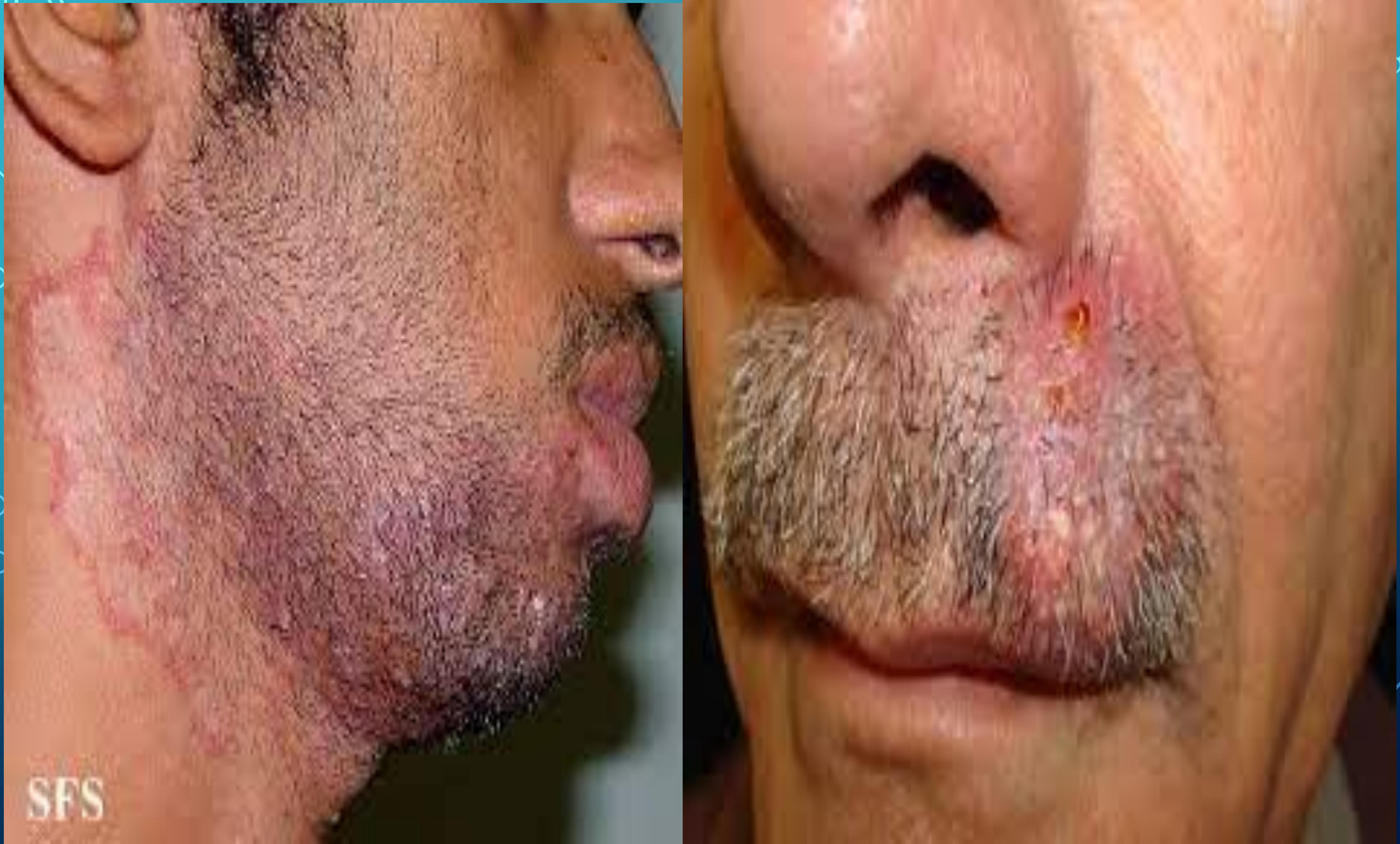


TINEA BARBAE

- Dermatophyte infection of the beard and Mustache in males
- Inflammatory : due to involvement of the hair follicle (pain) and skin (itching).
- Slowly progressive, farmers(violceum,mentographytes, rubrum)
- DDx: bacterial folliculitis
- Systemic treatment is the role


SFS

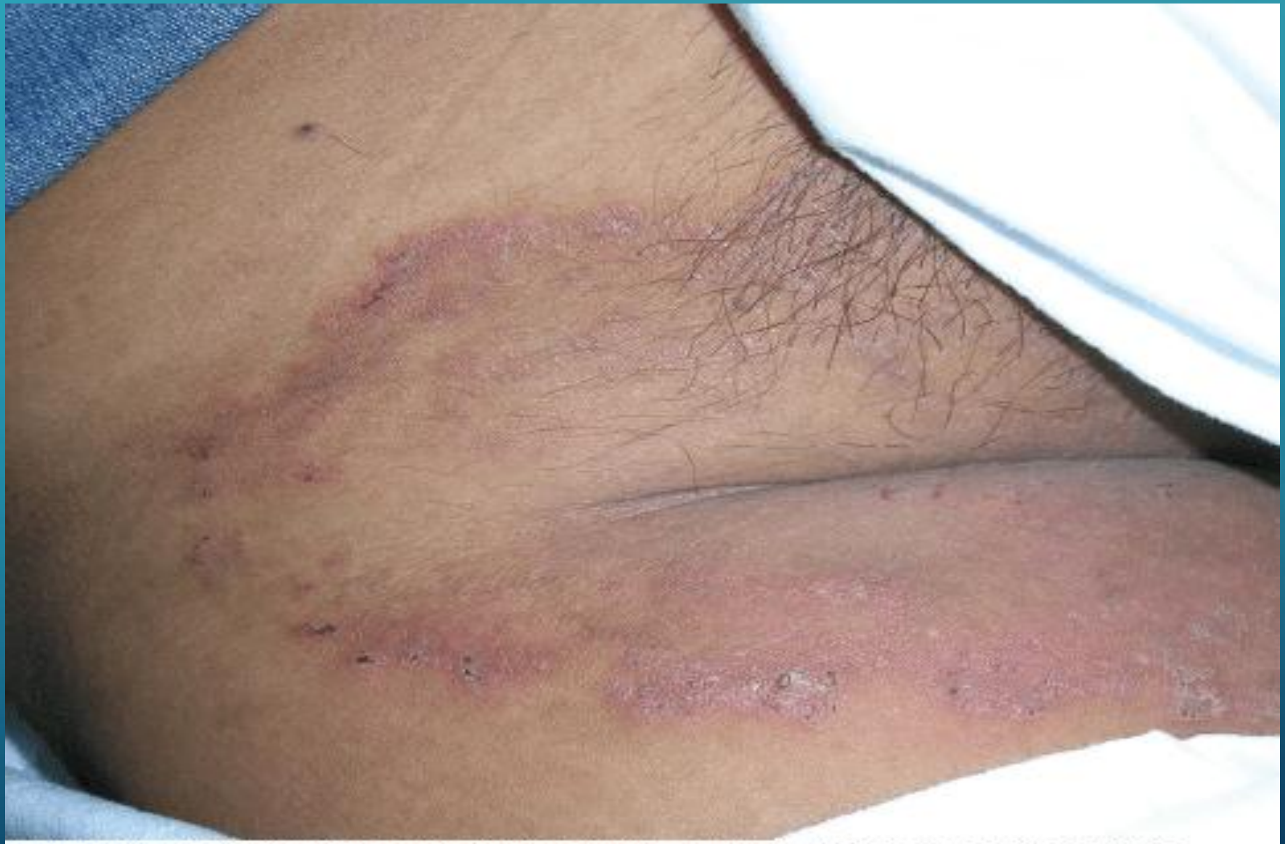
TINEA CRURIS (JOCK ITCH)

- Almost exclusively in post pubertal males/ RF
- Itching increases with moist climate, bilateral
- DDx: Intertrigo

- Inverse psoriasis

- Erythrasma

TINEA CRURIS (JOCK ITCH)



Source: Richard P. Usatine, Mindy Ann Smith, Heidi S. Chumley, Camille Sabella, E.J. Mayeaux, Jr., Elumalai Appachi: *The Color Atlas of Pediatrics*: www.accesspediatrics.com

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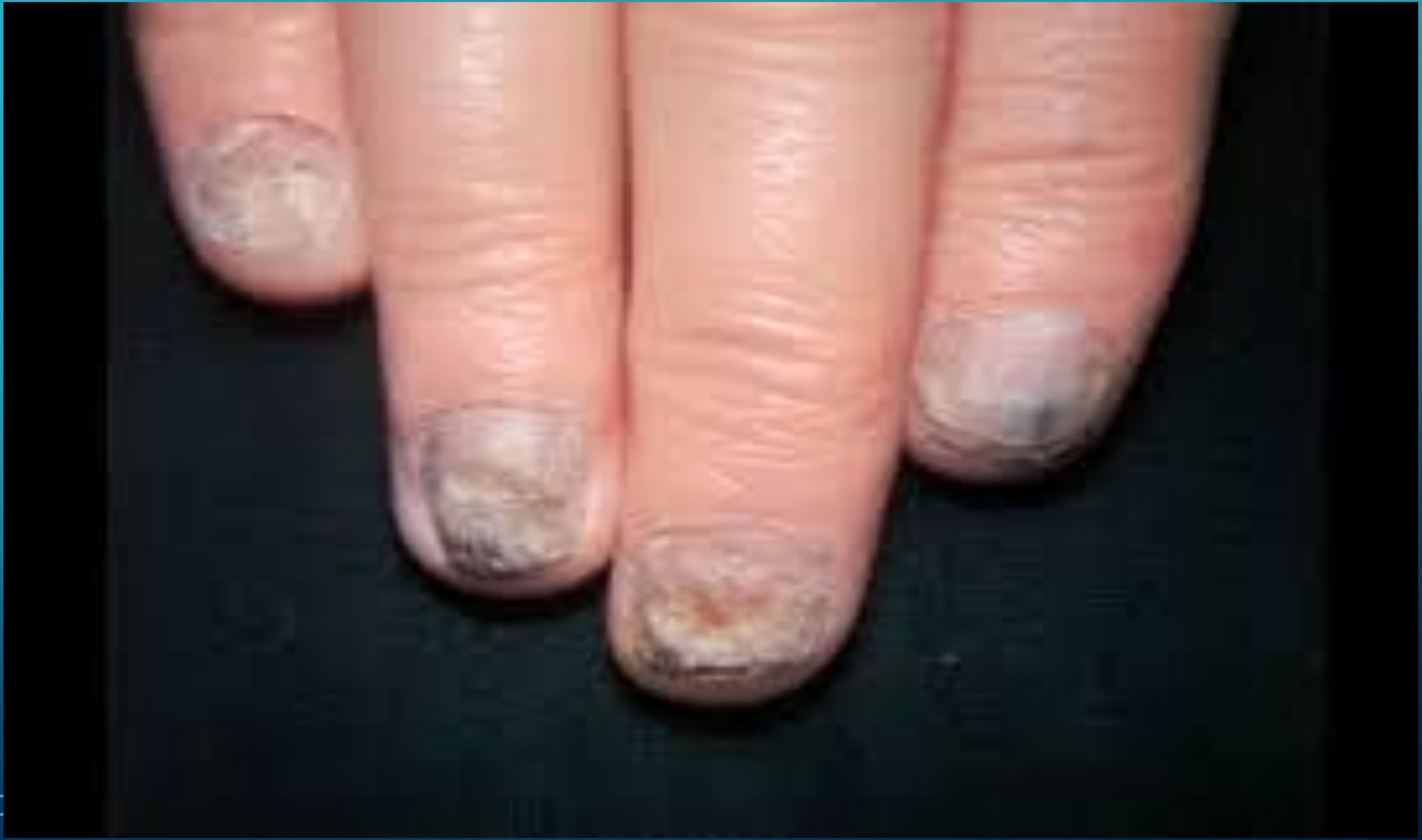
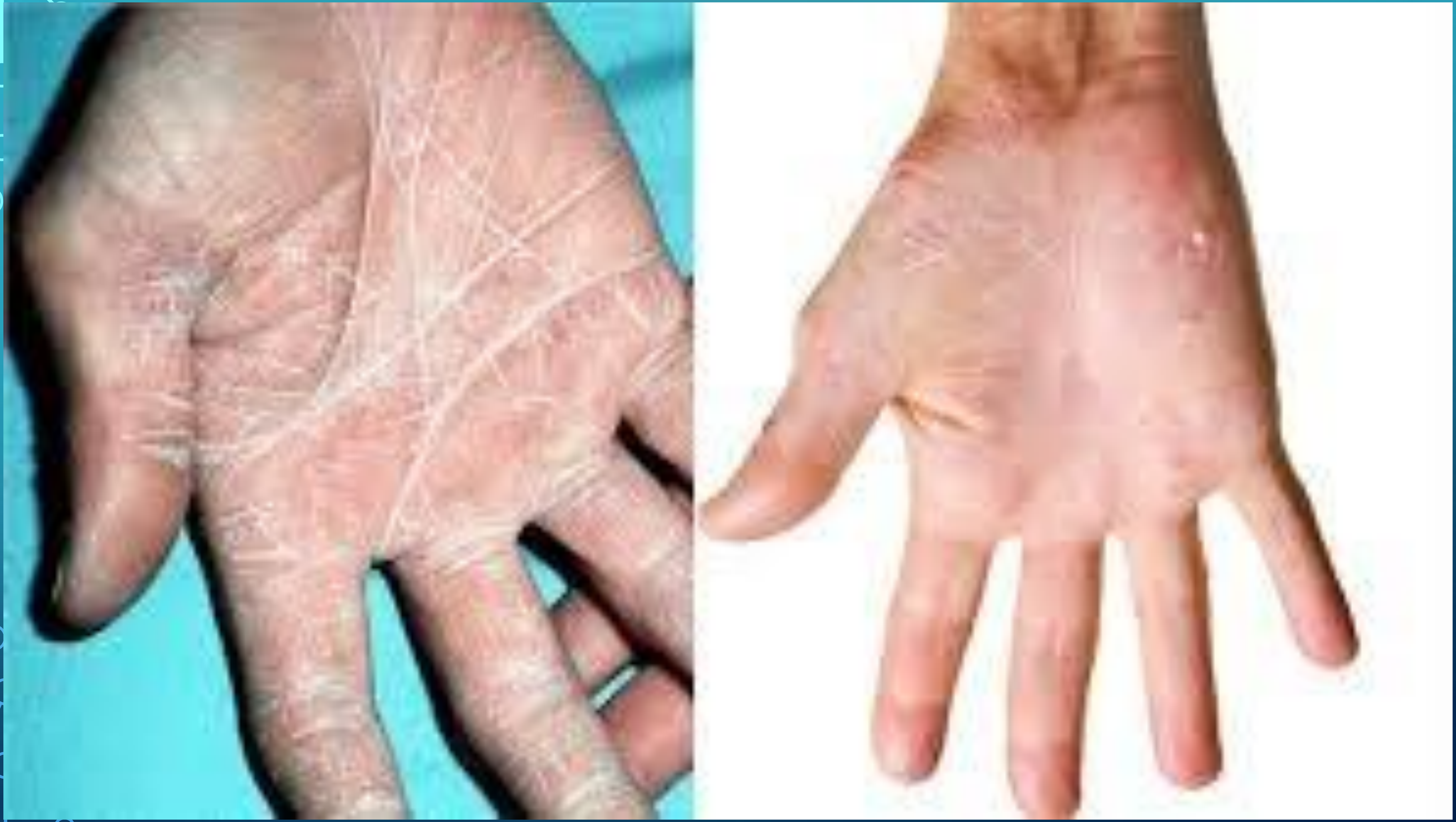
ONYCHOMYCOSIS (UNGUIUM)

- Tinea of the nails
- Prolonged systemic ttt



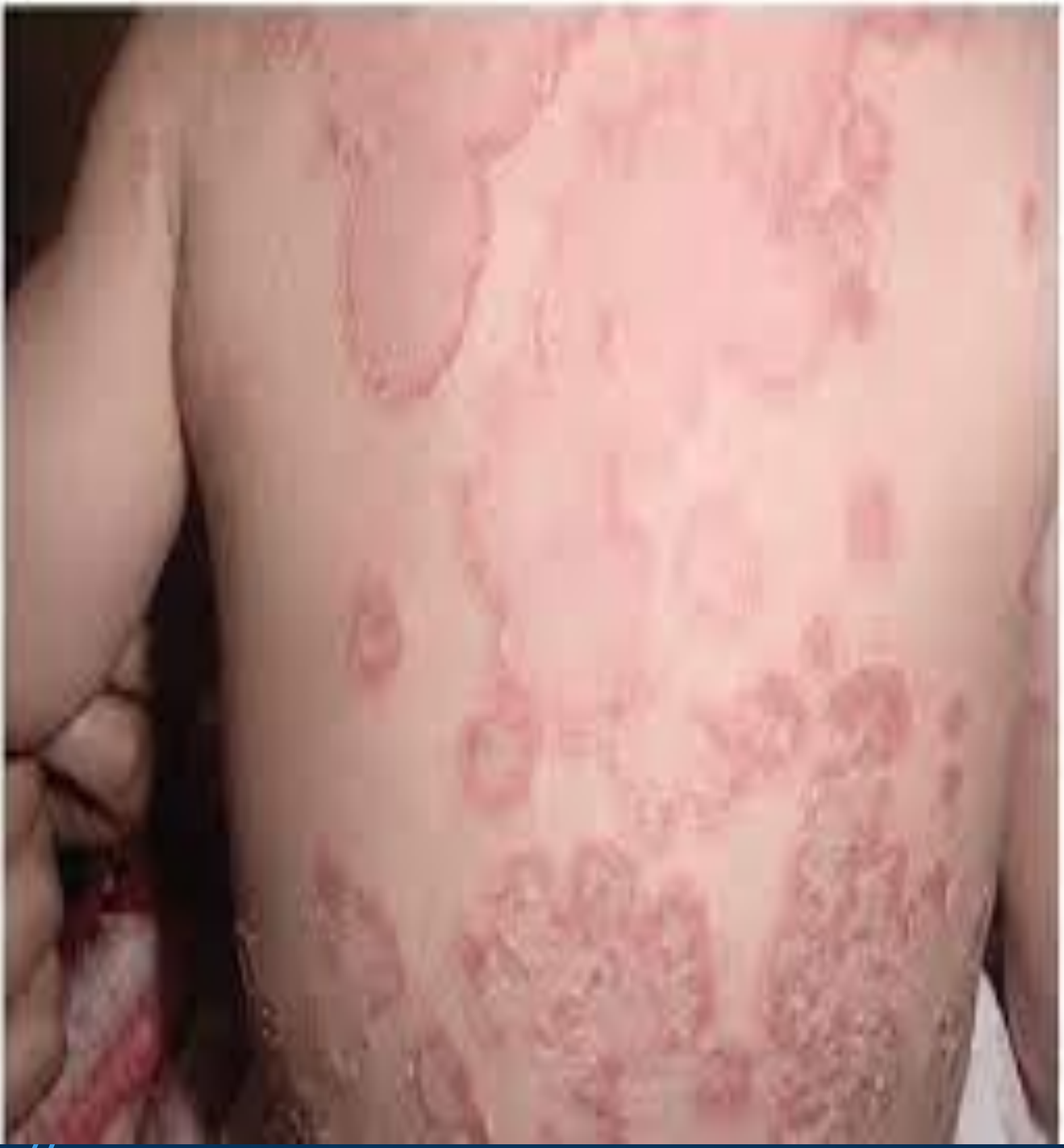
TINEA MANNUM

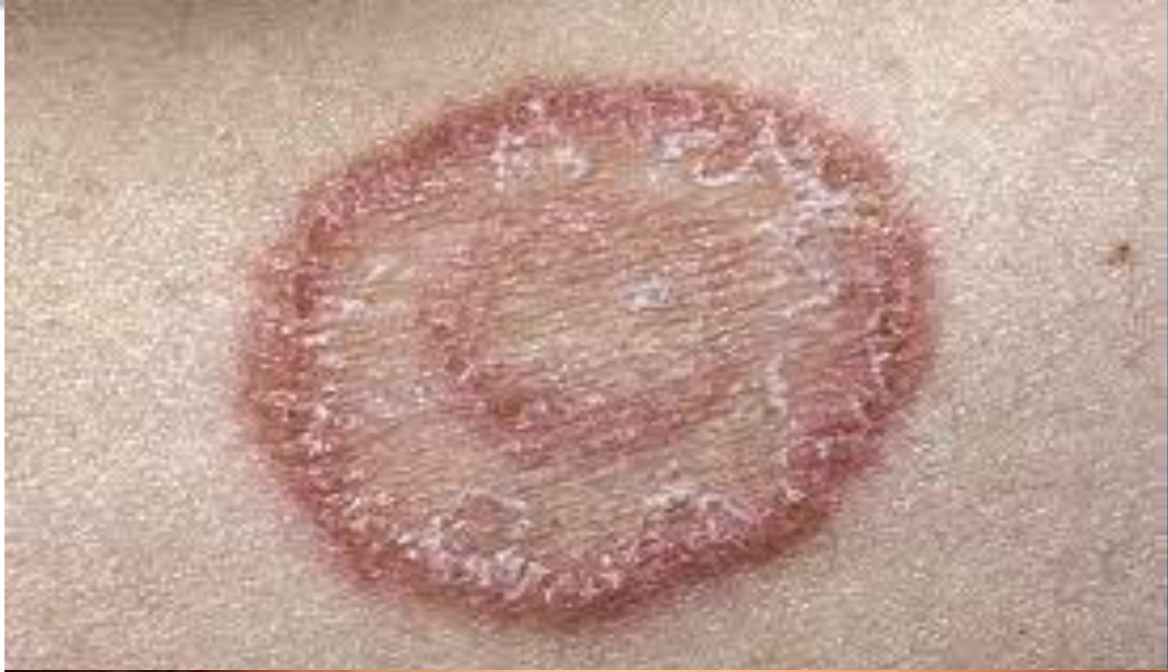
- Chronic , pruritic with fissures and pain
- Well demarcated scaling patches, hyperkeratosis, scaling , central clearing
- DDx:
- Atopy, CD, Lichen simplex chronicus,
- Better to use systemic treatment .

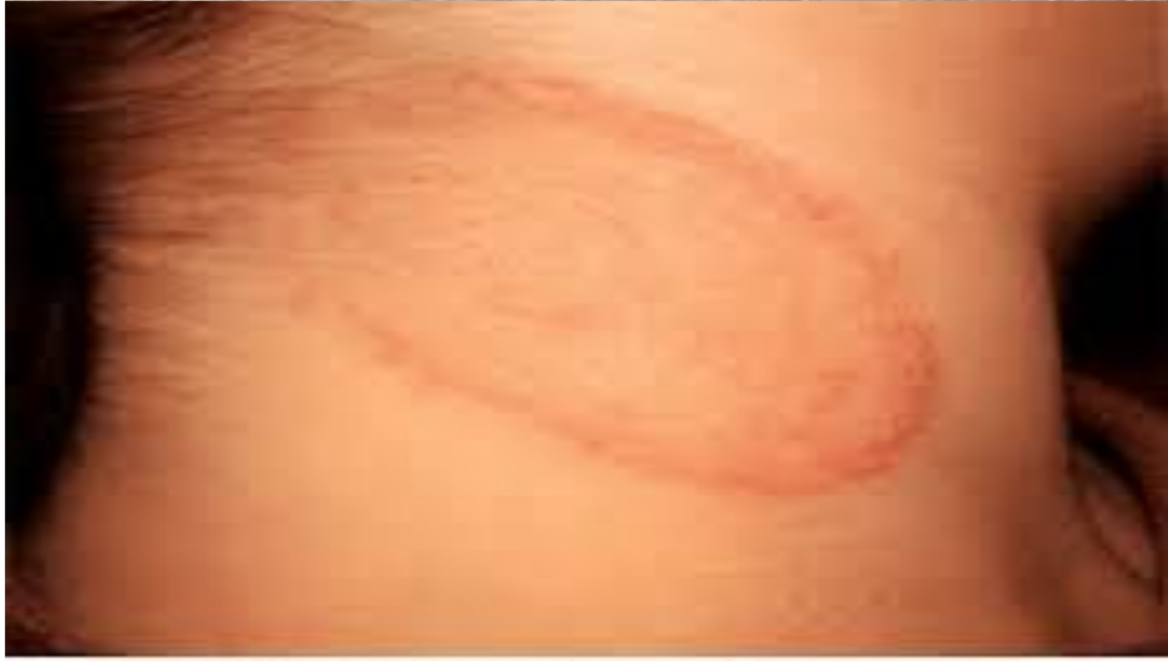


TINEA CORPORIS:

- Dermatophyte infection of the trunk, legs, arms excluding the face, groin, hands and feet.
- Appears in exposed skin as circinate, single or multiple itchy lesion.
- Sharp margin (raised edge =active border) with minute papules and pustules with a clearing center







Source: Neil S. Prose, Leonard Kristal: Weinberg's
Color Atlas of Pediatric Dermatology, Fifth Edition
www.accesspediatrics.com

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TINEA CORPORIS:

- DDx:

- Circinate impetigo

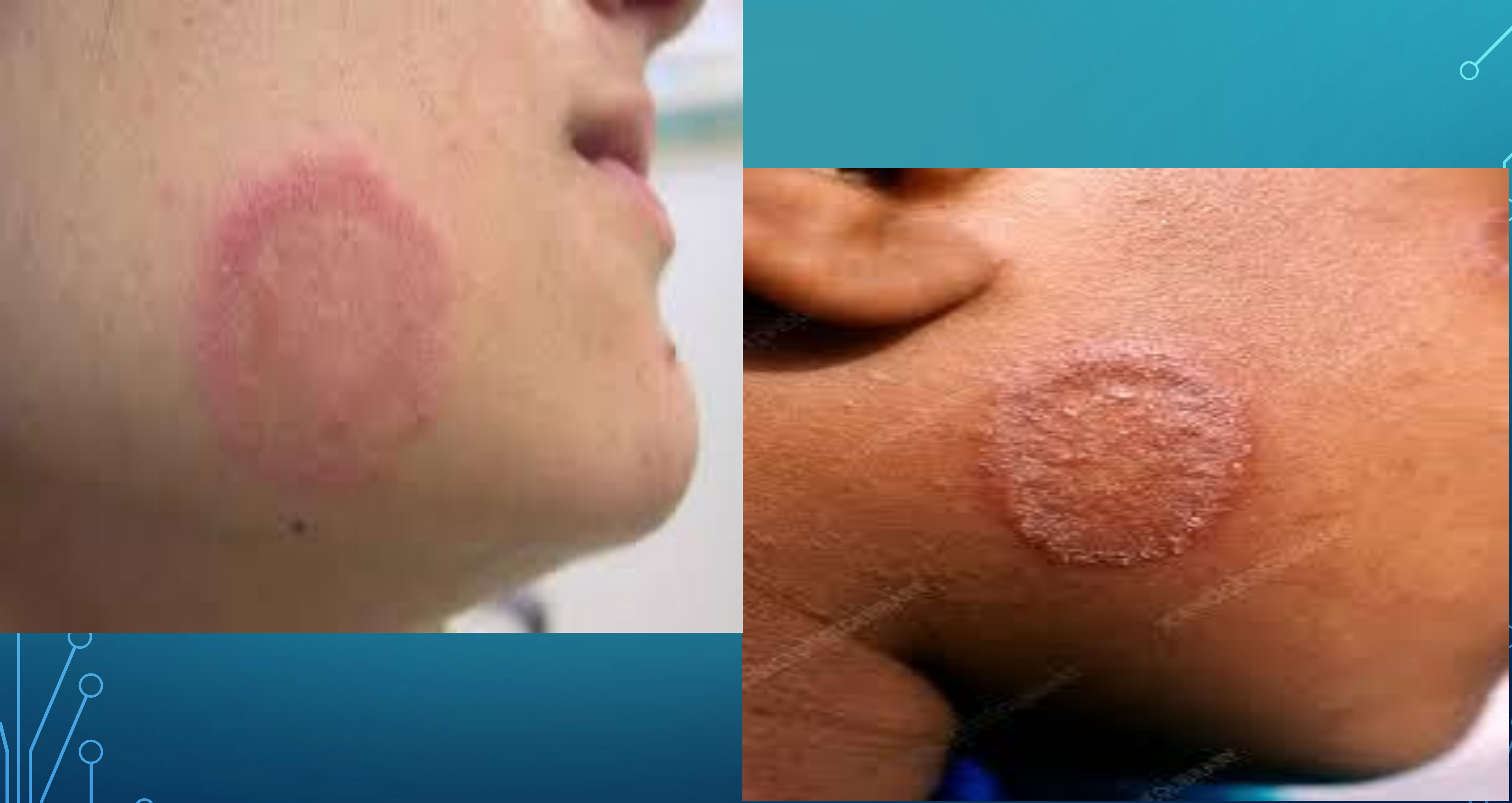
- Pityriasis rosea

- Pustular psoriasis

- Discoid eczema

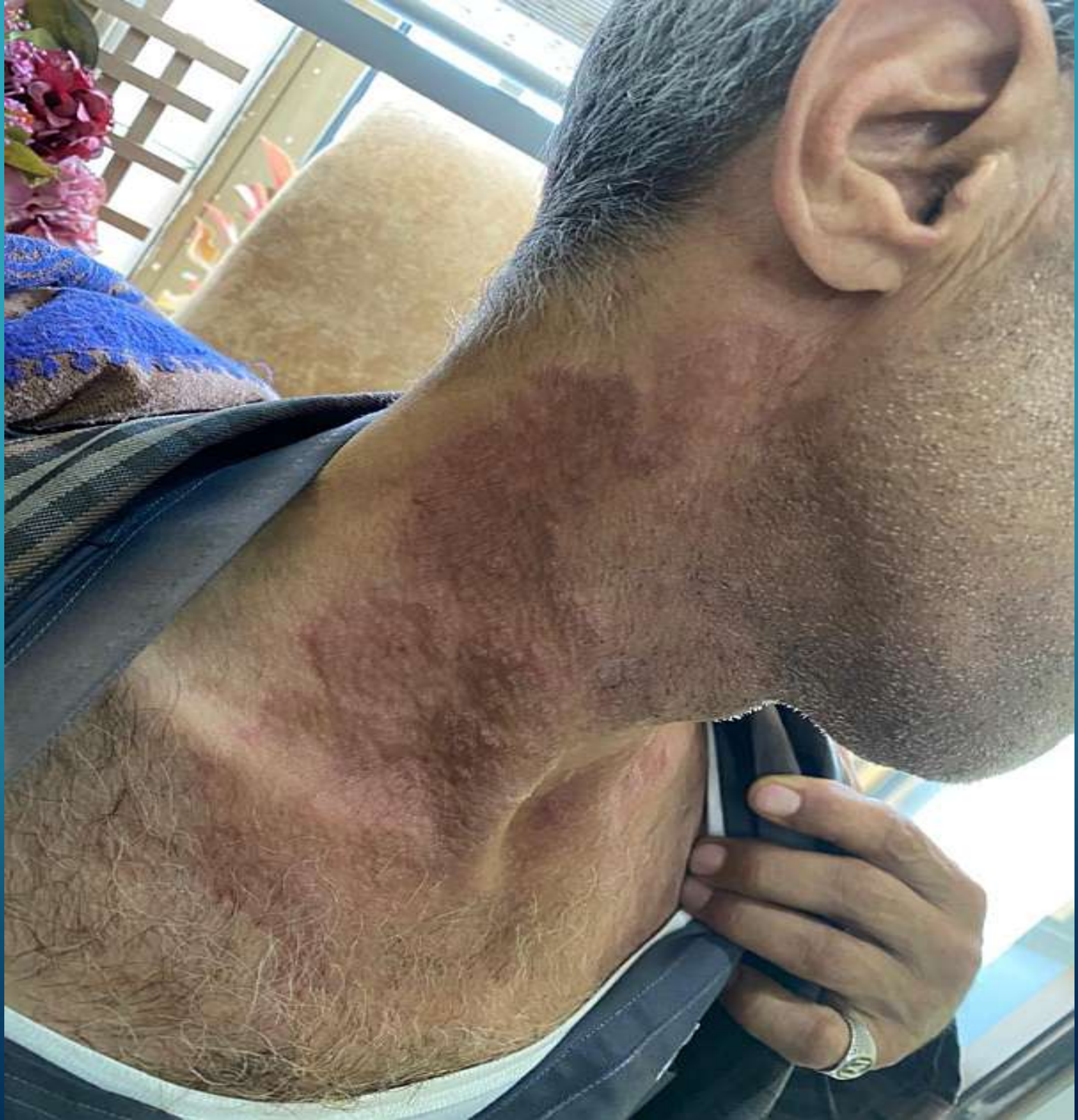
TINEA FACIALS

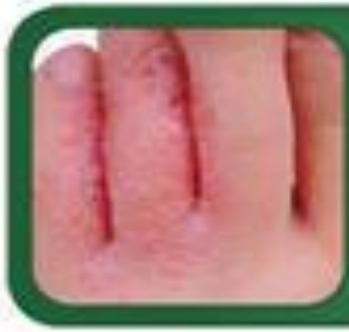
- Located in the face
- Well circumscribed, itchy, erythematous scaly macule or patch
- R/F: animal exposure, chronic CS exposure.
- DDx: seborrheic dermatitis, CD,



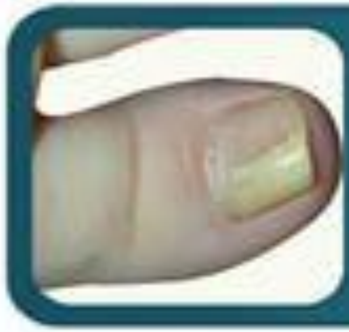
TINEA INCOGNITO:

- Associated with folliculitis.
- Occurs after application of topical corticosteroids
- Lesions vary from asymptomatic to pruritic and painful.
- Systemic antifungal may be prescribed.

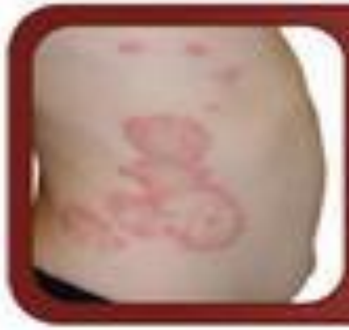




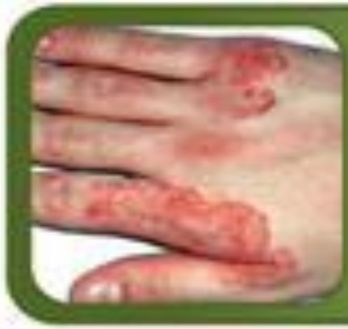
Tinea pedis :
Also known as athlete's foot, it affects the feet



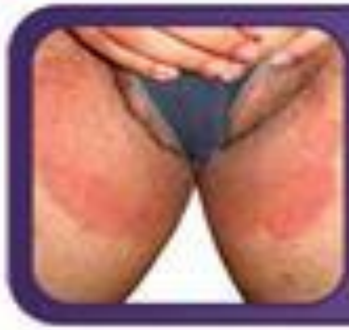
Tinea unguium:
Ringworm of the fingernails and toenails



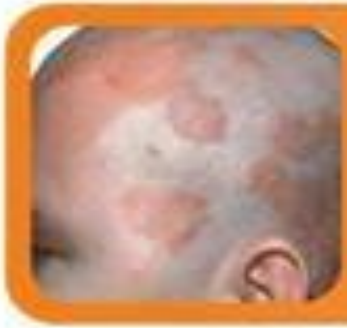
Tinea corporis:
It affects the arms, legs and trunk



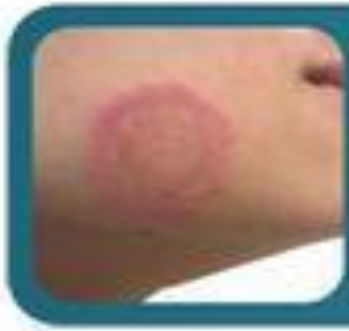
Tinea manum:
Ringworm of the hands and palms



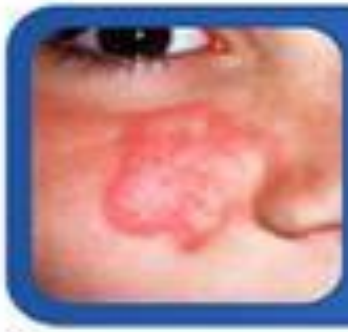
Tinea cruris:
Known as Jock Itch, this affects the groin area



Tinea capitis:
Ringworm affecting the scalp



Tinea barbae:
Ringworm of facial hair (beard)



Tinea faciei:
Also known as face fungus, it affects parts of the face

PITYRIASIS VERSICOLOR

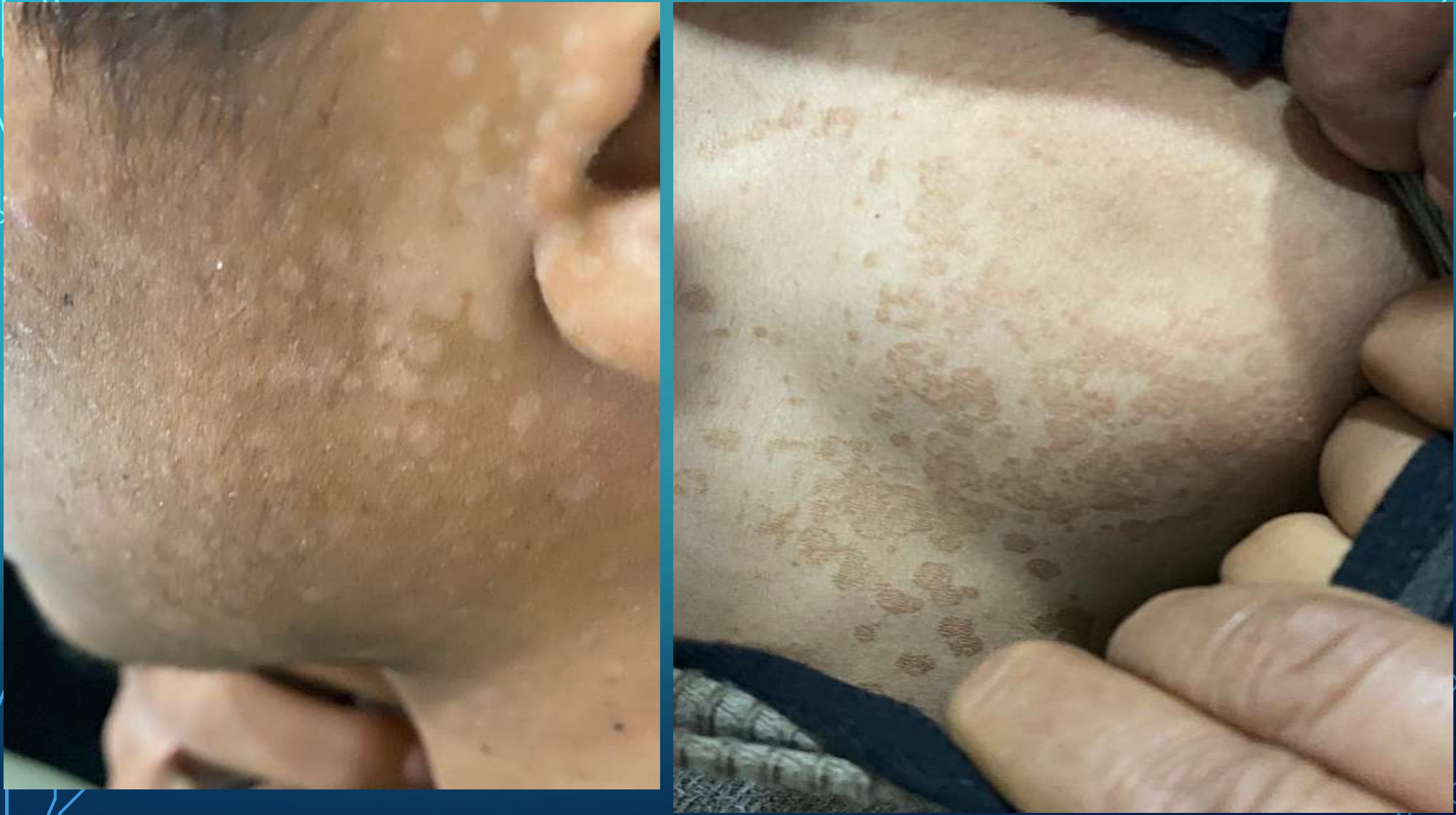
- Caused by overgrowth of Malassezia furfur
- Chronic asymptomatic scaley disorder.
- Well demarcated scaley patches: hyper pr hypopigmented
- Most commonly on the trunk
- Not contagious

- R/F;

- Warm seasons, tropical climate
- Hyperhidrosis , exercise
- Oily skin
- Corticosteroids ttt
- Immunodeficiency

- Additional ttt:

- Selenium sulfide
- Sulphur
- Salicylic acid



CANDIDIASIS:

- Most commonly caused by candida albicans
- Superficial infection of the skin and MM.
- Affects moist and occluded cutaneous surfaces

CANDIDIASIS:

- Clinical Manifestation:
- Intertrigo
- Diaper dermatitis
- Interdigital
- Follicular candidiasis
- Paronychia (chronic).
- Vulvovaginitis,
- Angular cheilitis , moniliasis

CANDIDIASIS:

- Clinically:

- Satellite lesions
- Initial pustules on erythematous base that becomes eroded and confluent.
- Inframammary, axillae and groin.
- Managemnt : prevention, oral and topical antifungals

TREATMENT OF FUNGAL INFECTION

1) Topical antifungal preparations:

- Imidazole group : miconazole, ketoconazole, clotrimazole
- Allylamines: Terbinafine , naftifine

2) Systemic treatment:

- Terbinafine: 250 mg tab /day
- Itraconazole : 100 mg cap, oral solution 10-40 mg /kg

TREATMENT OF FUNGAL INFECTION

- Fluconazole: 100-150 mg
- Ketoconazole
- Griseofulvin:12,5 mg /kg : micronized and ultra micronized

THANK YOU

PAPULOSQUAMOUS DISEASES

DR. SAHAR AHMED AL-AWDI

Dermatology department –Faculty of medicine

Sana'a university

PAPULOSQUAMOUS DISEASES

1- Psoriasis

2- lichen planus

3- Pityriasis Rubra pilaris

PSORIASIS

- Papulosquamous chronic skin disease with unpredictable remissions and relapses.
- Mostly affecting adults (14-40 years).
- Incidence: 1-2% of population

PROACTIVE FACTORS:

1- Genetic: Polygenic inheritance(PSORS1), 30% have positive family history, increases with HLA ass B13,BW17, CW6,B27

2- Trauma: Koebner phenomena

3- Infection: Streptococcal infection

4- Weather: Worse in winter

PROACTIVE FACTORS:

5- Endocrine: May exacerbate with puberty, menopause , pregnancy.

6- Drug induced: antimalarials, withdrawal of steroids, Lithium, propranolol.

7- Emotional stress: 40% of cases

8- Obesity , smoking , alcoholism

PATHOGENESIS:

- Stimulation: trauma, infection, …
- Secretion: IL1, IL8, TNF alpha, interferon gamma, IL 2.
- Response: epidermal hyperproliferation (4-5 day) , parakeratosis, Scale formation.
- Capillary dilatation: upper dermis (Auspitz sign)
- Accumulation of inflammatory cells: Neutrophils (microabcesses)

CLINICAL PRESENTATION:

- Scaling, and erythematous plaques that may be painful and/or often severely pruritic (QoL)
- Sharply demarcated, erythematous papules and plaques often covered with silvery-white scales
- If the covering scale is removed, a salmon-pink to erythematous lesion is exposed, usually with punctuate bleeding (Auspitz sign)

PSORIASIS VARIANTS:

Morphological variants:

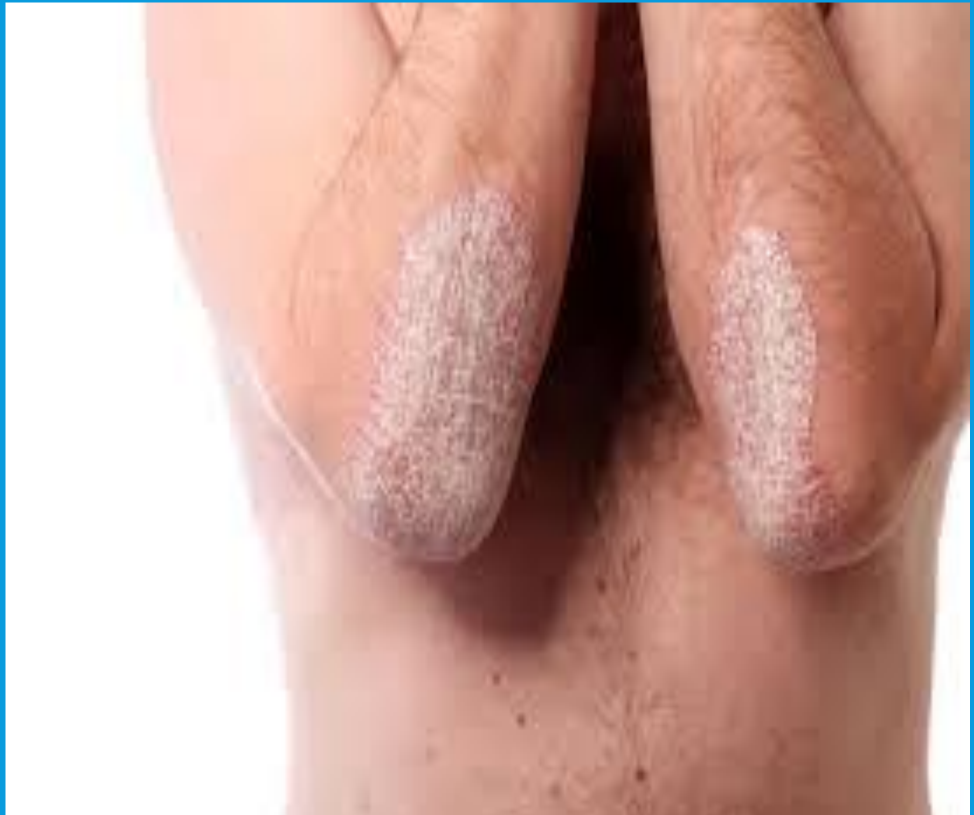
- Plaque psoriasis
- Guttate psoriasis
- Geographical psoriasis.
- Follicular
- Annular.
- Erythrodermic psoriasis

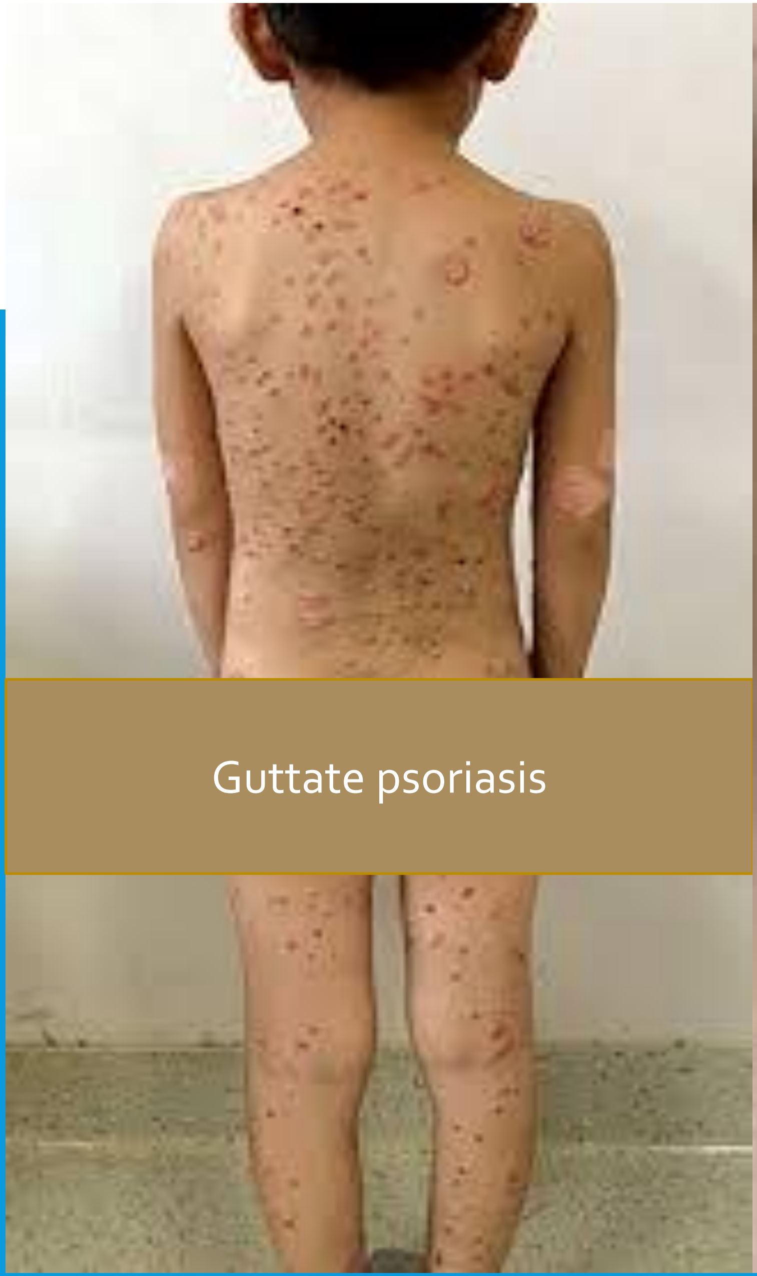
- Psoriasis Vulgaris
- Scalp Psoriasis
- Nail Psoriasis
- Pustular psoriasis
- Erythrodermic psoriasis
- Inverse Psoriasis
- Psoriatic arthritis…..

CLINICAL PICTURE:

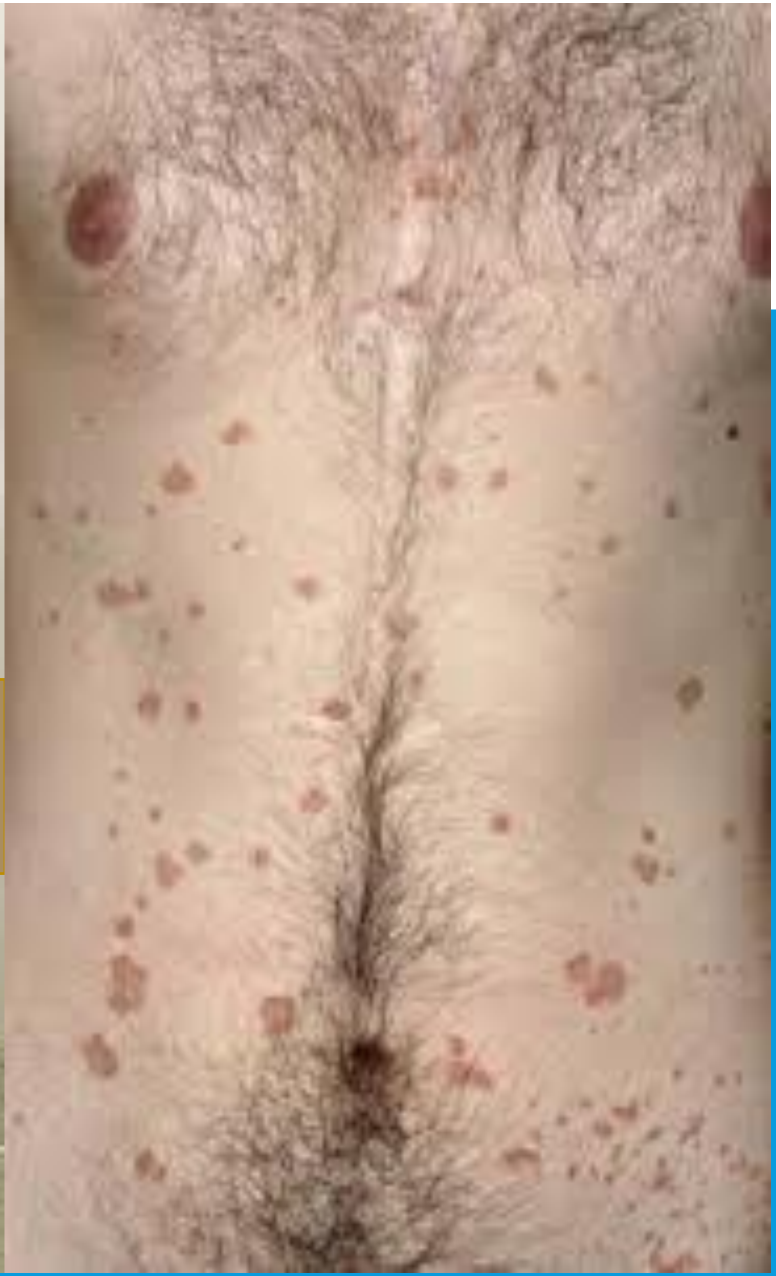
Morphological patterns:

1- Plaque Psoriasis (classical type): the most common type affecting around 85% of psoriatic patients.





Guttate psoriasis



2- GUTTATE PSORIASIS:

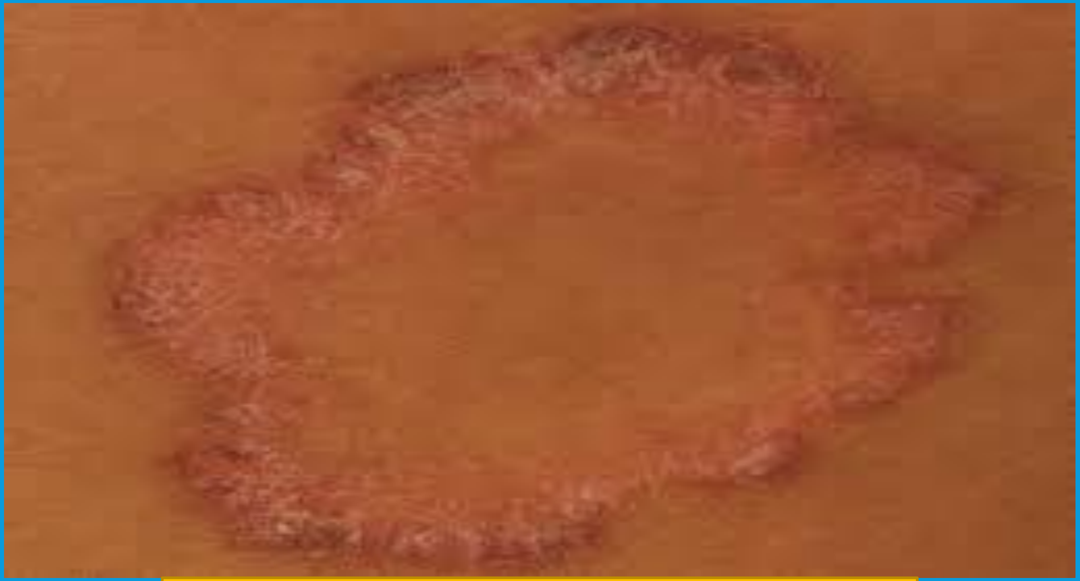
Lesions are the size of drops of water (may follow streptococcal infection).

Most common variant in children.

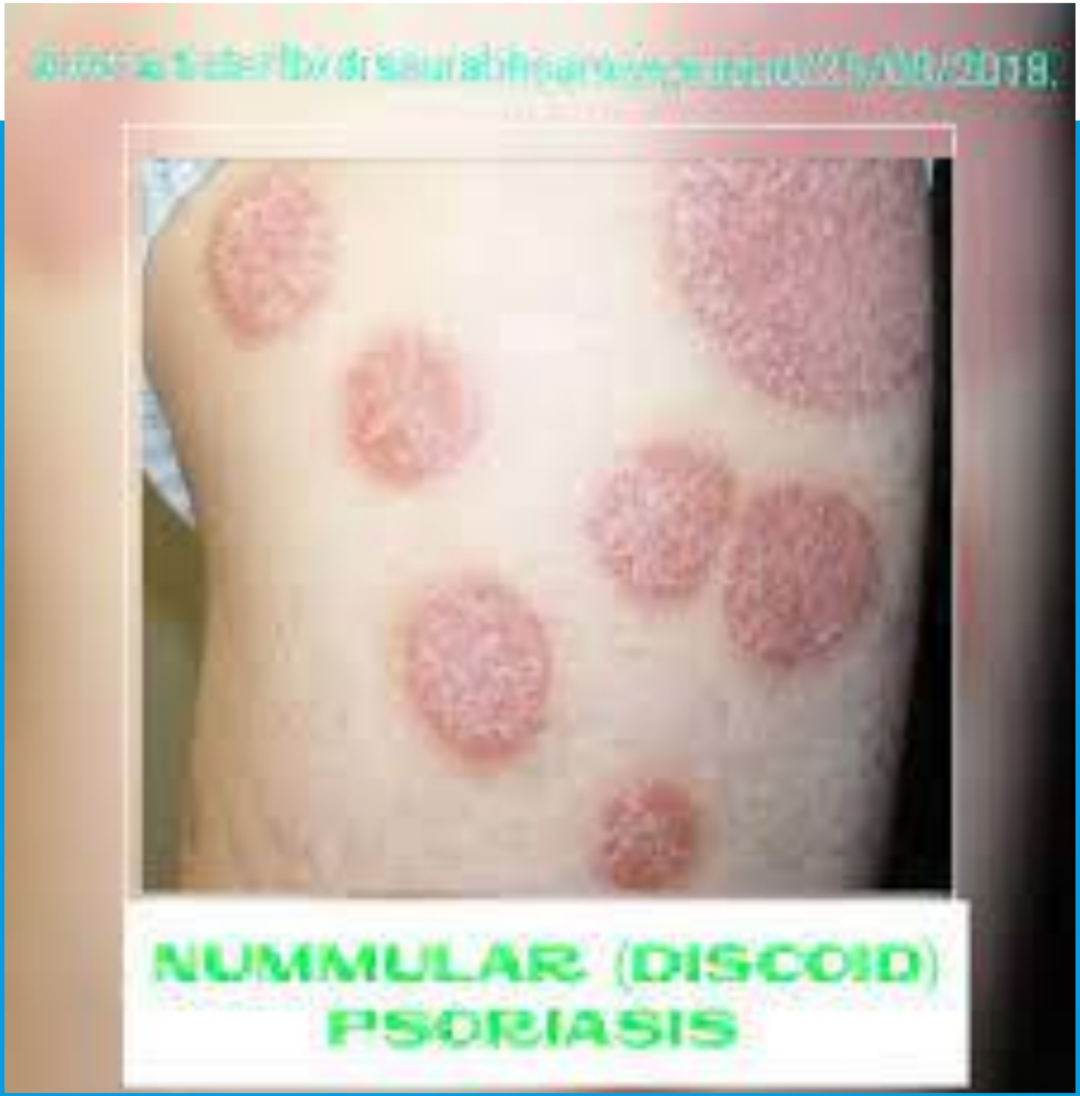
MORPHOLOGICAL PATTERNS:

3- Discoid/annular psoriasis:

Coin shaped lesions



Annular psoriasis


NUMMULAR (DISCOID)
PSORIASIS

MORPHOLOGICAL PATTERNS:

Geographical psoriasis: curved large lesions in the back

Follicular psoriasis: adult females and children located in the thighs

Linear psoriasis : Koebner's phenomena

MODIFICATION BY SITE

1- Scalp psoriasis: band like lesions in the anterior border of hair line

2- Flexural psoriasis(inverse): Affects flexures(sub mammary, groin, axillae),itching , redness and no scales.

3- Palmoplantar psoriasis:

- Pustulosis
- Thick fissured plaques
- Typical silvery plaques
- Sole rupioid form

MODIFICATION BY SITE

4- Nail Psoriasis: pitting , ridges , grooves , onycholysis , subungual hypertrophy

5- Psoriatic arthropathy: rheumatic factor negative arthritis

6- Erythrodermic psoriasis: Precipitated by infection, hypocalcemia, antimalarials , CS withdrawal(ER); sparing of face , 90% of body involvement, leukocytosis, ↑ESR,

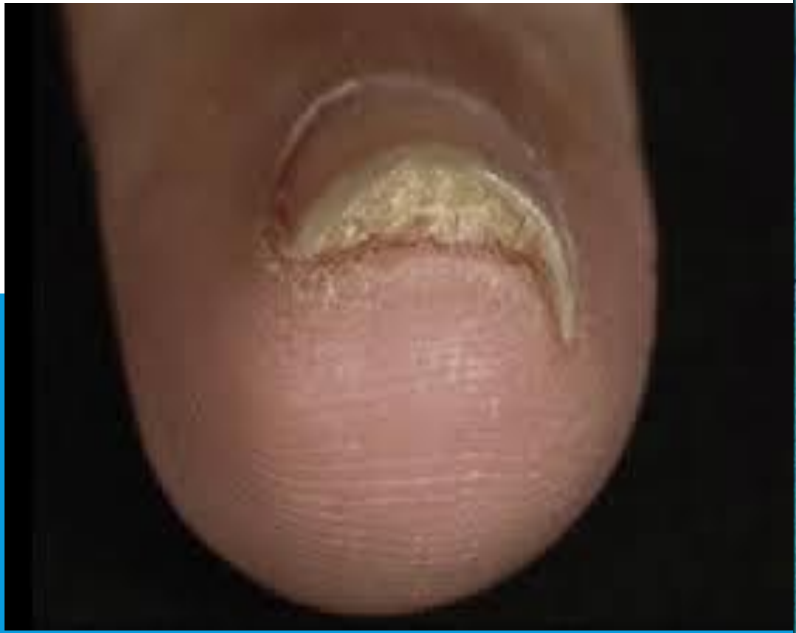
MODIFICATION BY SITE

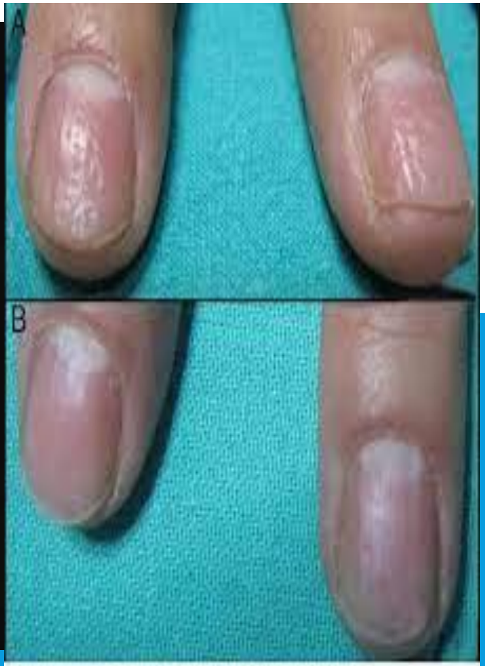
7- Sebopsorisis:

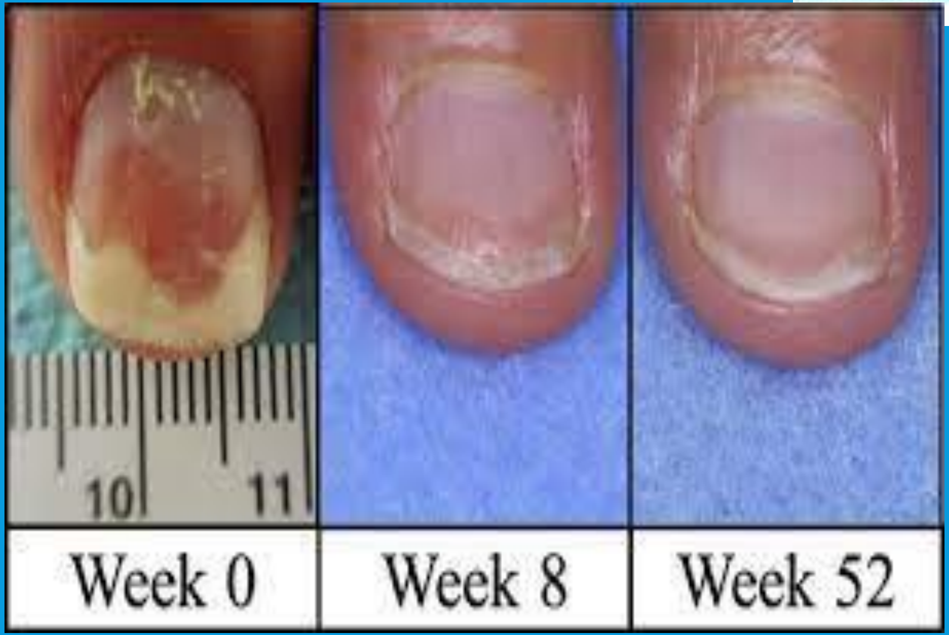
Usually, seborrheic distribution

8- Mucosal lesions: more in pustular psoriasis

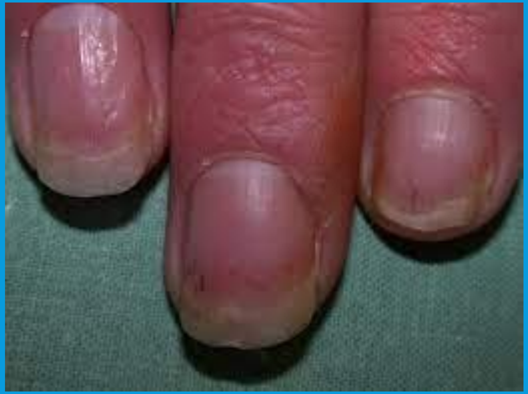
9- Pustular psoriasis: precipitated by irritating topicals , hypocalcemia and pregnancy.







Week 0 | Week 8 | Week 52



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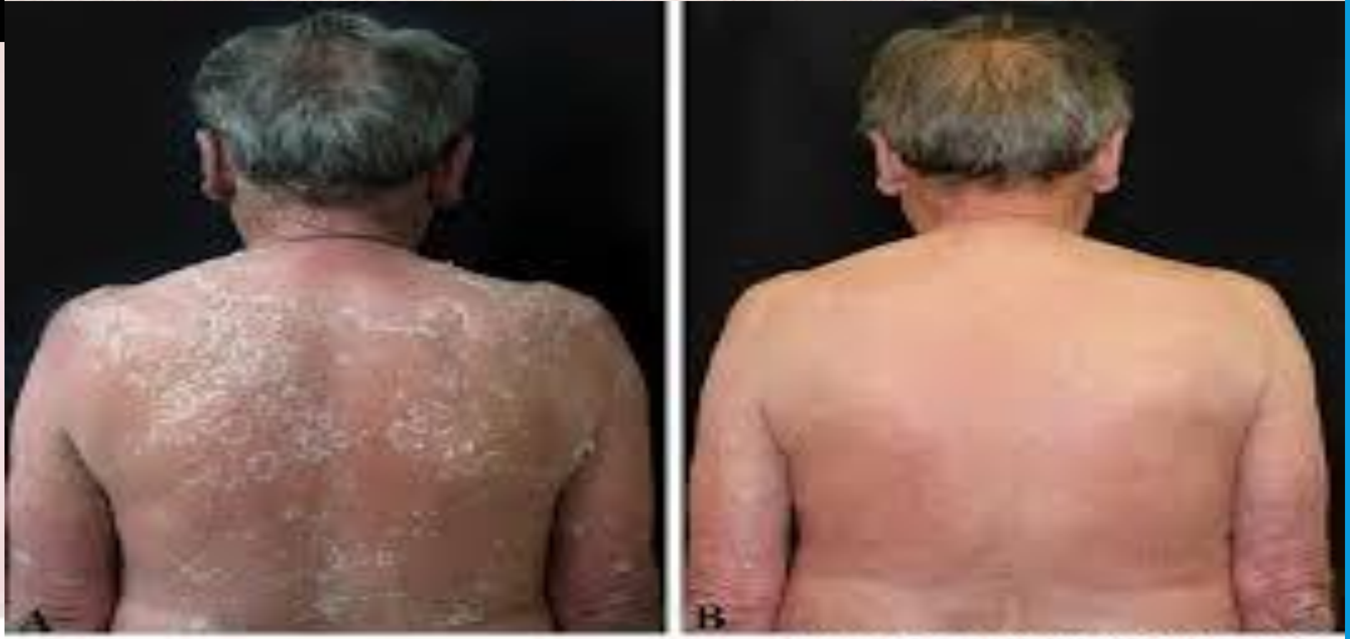

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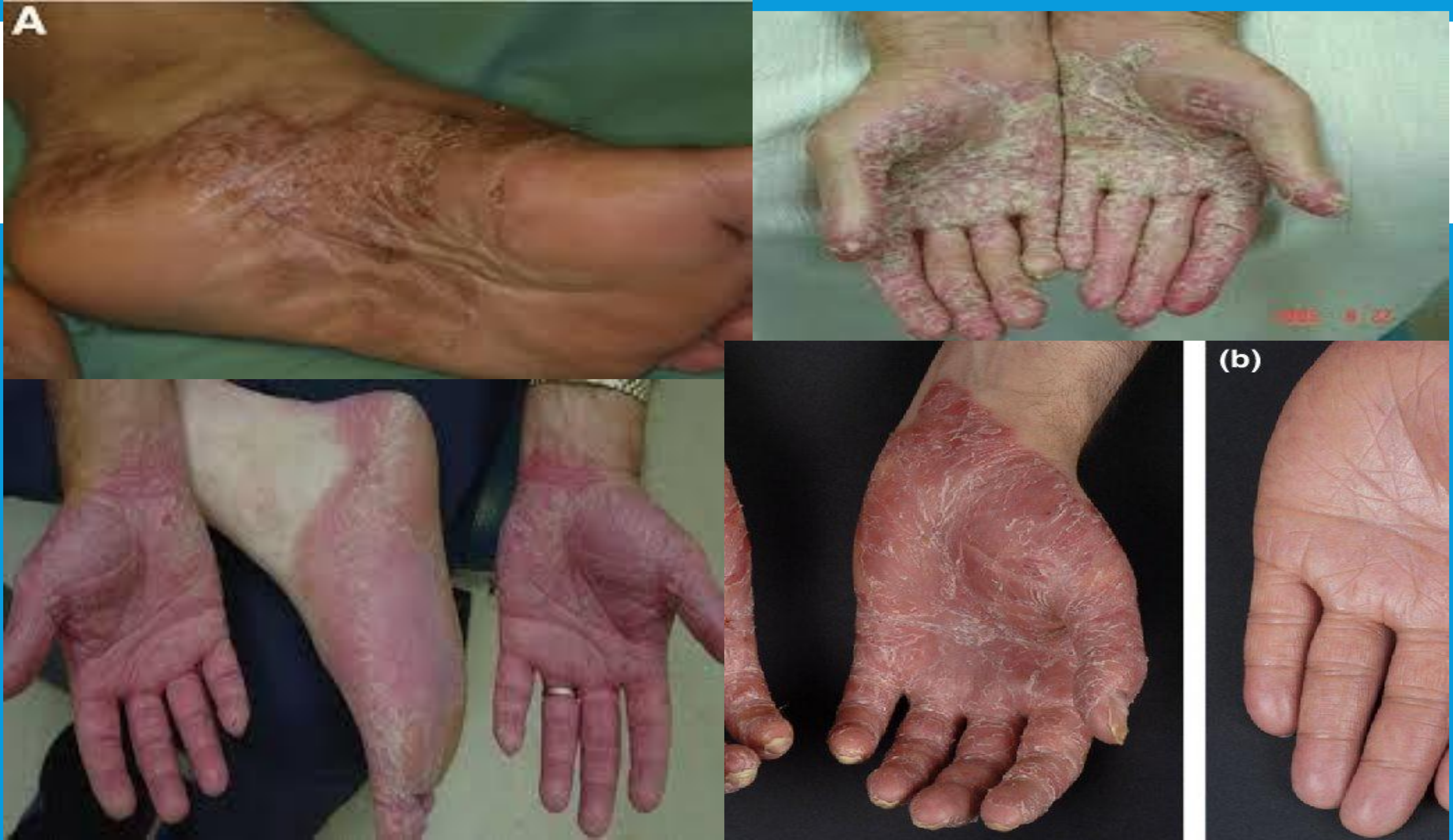
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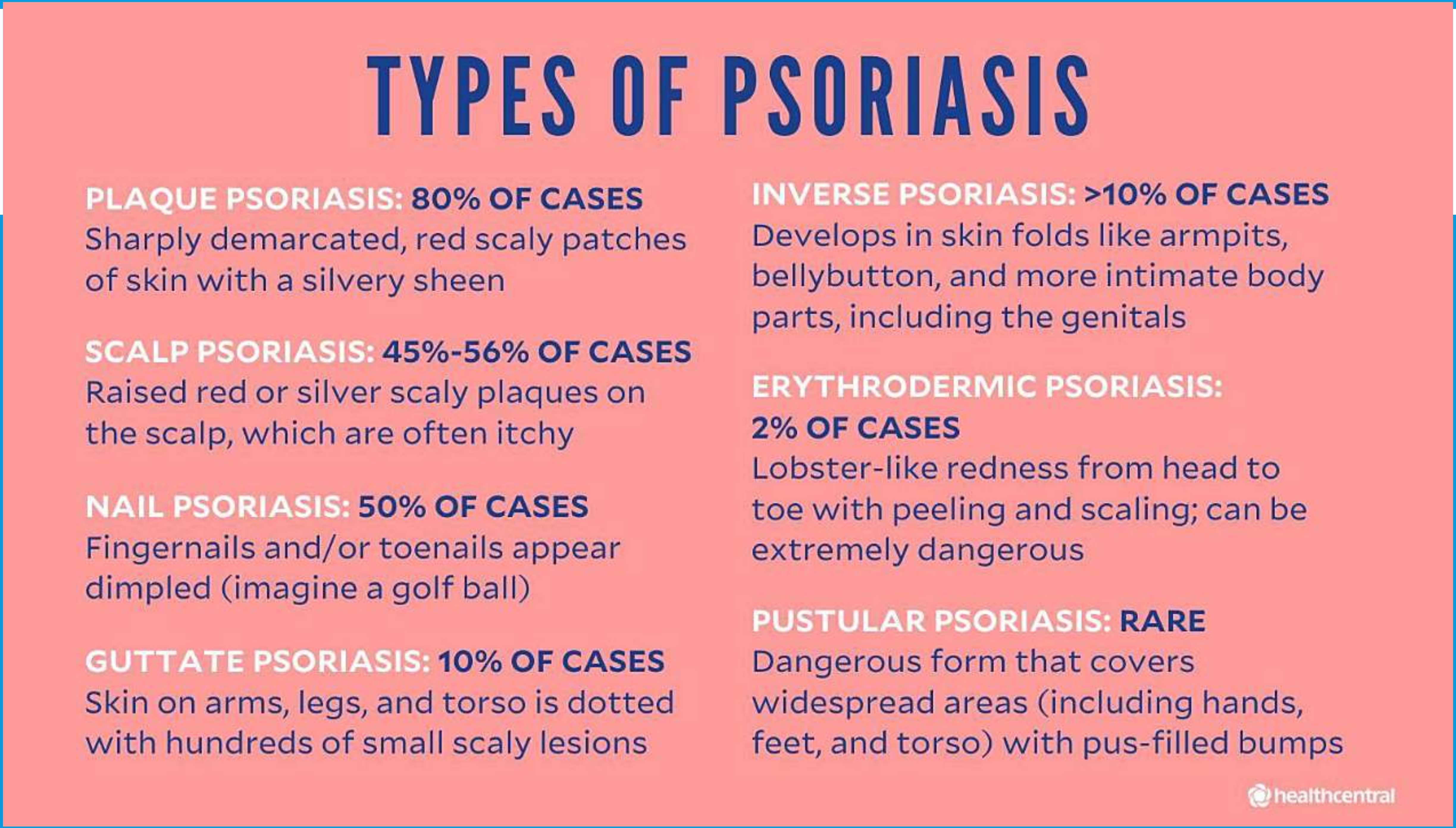


TYPES OF PSORIASIS

PLAQUE PSORIASIS: 80% OF CASES
Sharply demarcated, red scaly patches of skin with a silvery sheen

SCALP PSORIASIS: 45%-56% OF CASES
Raised red or silver scaly plaques on the scalp, which are often itchy

NAIL PSORIASIS: 50% OF CASES
Fingernails and/or toenails appear dimpled (imagine a golf ball)

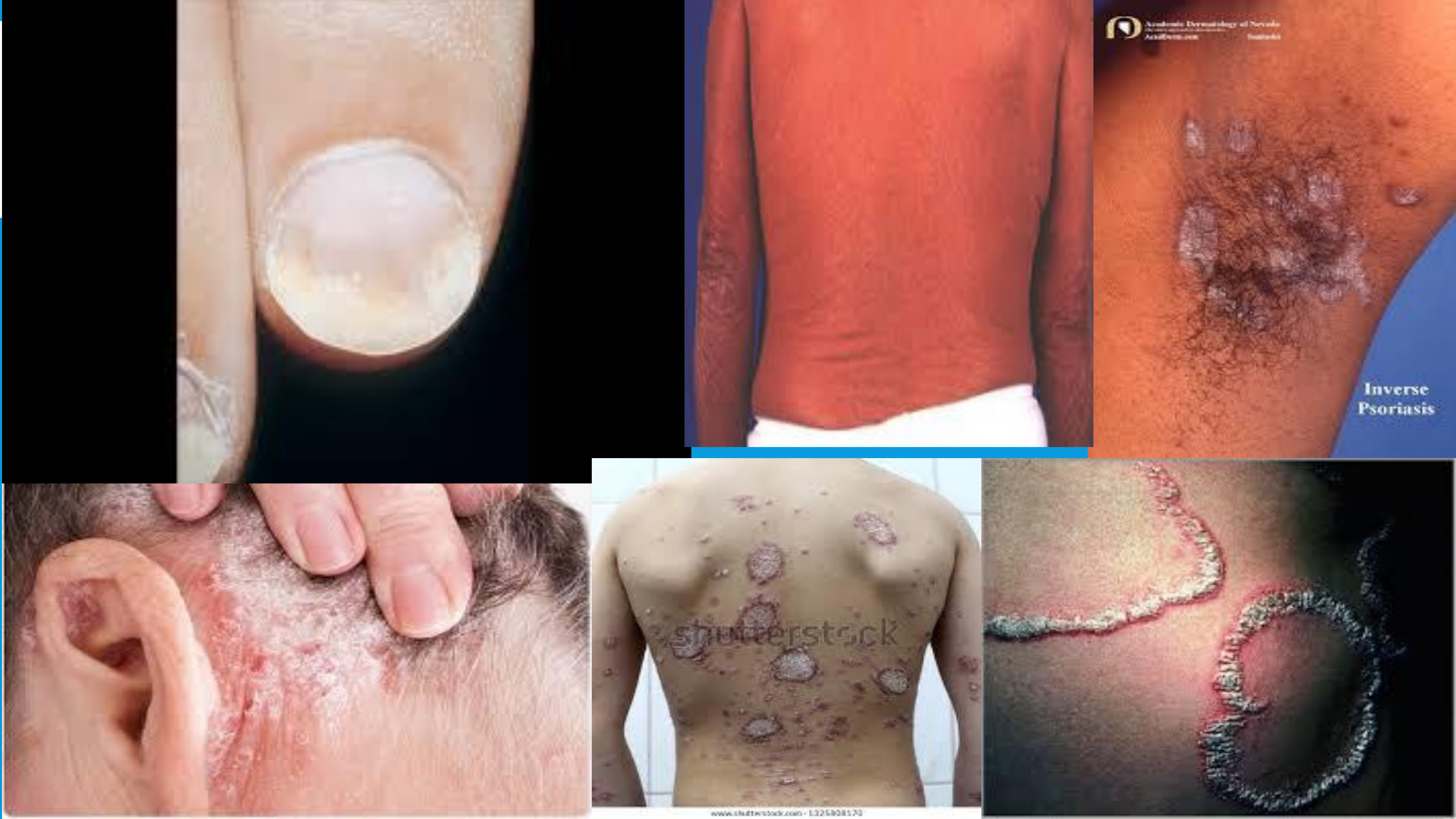
GUTTATE PSORIASIS: 10% OF CASES
Skin on arms, legs, and torso is dotted with hundreds of small scaly lesions

INVERSE PSORIASIS: >10% OF CASES
Develops in skin folds like armpits, bellybutton, and more intimate body parts, including the genitals

ERYTHRODERMIC PSORIASIS: 2% OF CASES
Lobster-like redness from head to toe with peeling and scaling; can be extremely dangerous

PUSTULAR PSORIASIS: RARE
Dangerous form that covers widespread areas (including hands, feet, and torso) with pus-filled bumps

healthcentral


Academic Dermatology of Nevada
AcadDerm.com
Inverse
Psoriasis
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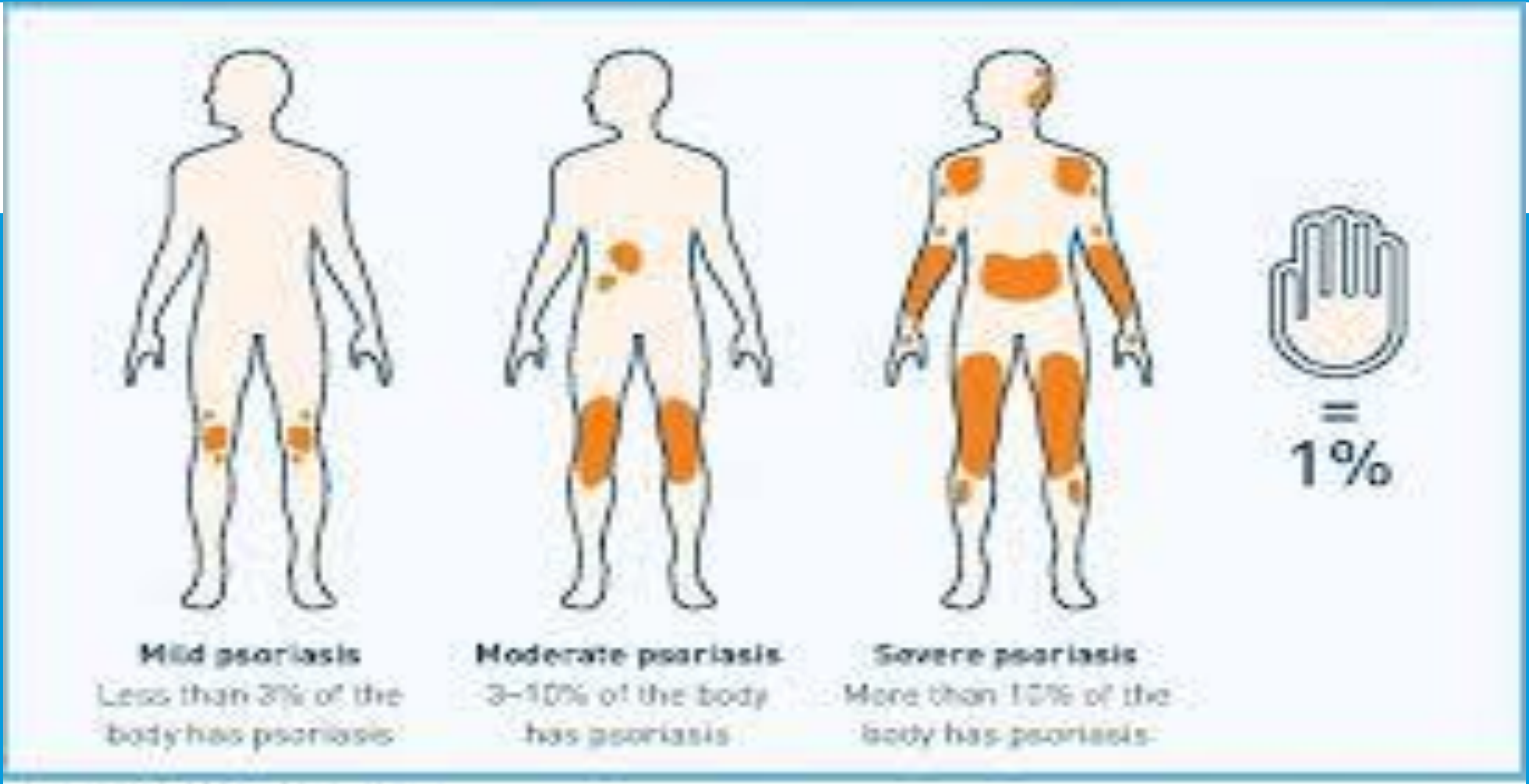
Mild psoriasis
Less than 3% of the body has psoriasis
Moderate psoriasis
3–10% of the body has psoriasis
Severe psoriasis
More than 10% of the body has psoriasis
= 1%

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TREATMENT OF PSORIASIS

A- Topical ttt:

1- Topical corticosteroids: mono or in combination

2- Calcipotriol: mono or in combination

3- Dithranol

4- Tar

5- Tazarotene

6- Salicylic acid 3%

TREATMENT OF PSORIASIS

B- Phototherapy: UVB(burn), NB UVB(311-313) , P(Psoralens)UVA (skin cancer)

C- Systemic treatment:

1- Methotrexate: BM suppression, indigestion, liver damage

2- Acitretin: teratogenic, lip dryness, hyperlipidemia

3- Cyclosporine: HTN, skin cancer, Renal toxicity

D- Biologicals: Alefacept, Efalizumab, Etanercept, adalimumab.

E- Immunological treatment: Ustekinumab

LICHEN PLANUS

- An idiopathic, chronic, papulosquamous, inflammatory, immune mediated skin disease that affects the skin, oral mucosa, genital mucosa, scalp, and nails.
- Lichen planus lesions have the six P's (planar [flat-topped], purple, polygonal, pruritic, papules, plaques); shiny.

LICHEN PLANUS

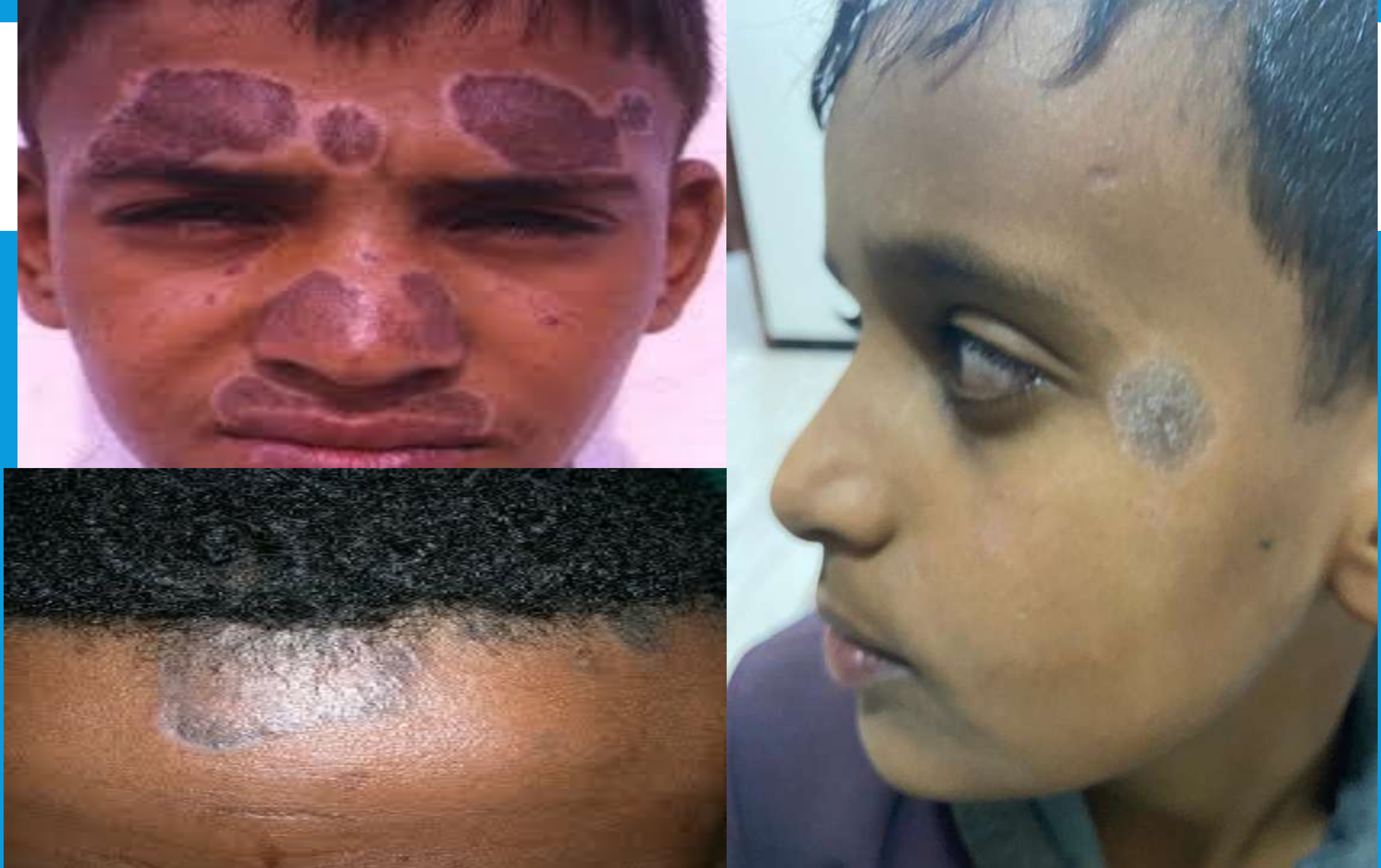
- Acute condition, affecting the flexor surfaces of the wrists, forearms, and legs, nails (10%) and MMs(70%). The lesions are often covered by lacy, reticular, white lines known as Wickham striae.
- Can occur at any site of the body Ch by Acanthosis, saw tooth thickening of the epidermis
- Affects 1% of adult population.

LICHEN PLANUS

- Oral lesions are common; 50-70%
- Nail lesions: 10%, nail ridging, grooving, striations , characteristic : pterygium formation. Fusion of the proximal nail fold with the nail bed
- Hair involvement: lichen planoplaris, can cause atrophy and permanent hair loss

CLINICAL VARIANTS OF LP

- Annular: glans, axillae and groin
- Actinic (tropicus): children and young adults.
- Oral erosive or ulcerative: painful, recalcitrant to ttt, HCV associated.
- Atrophic: may be due to resolution of LP lesions, at LLs mostly(lichen sclerosus et atrophicans).
- Lichen striates



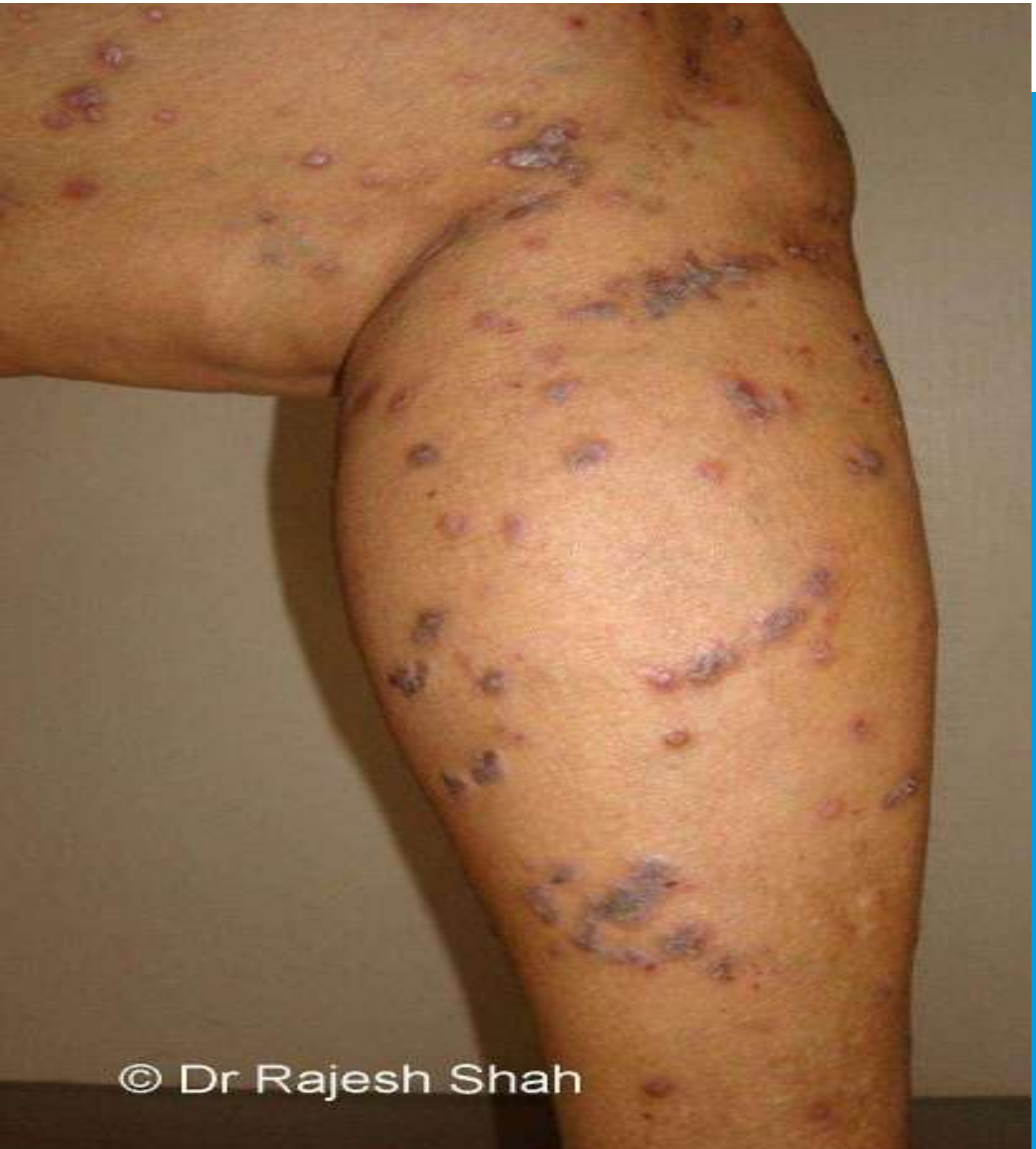
CLINICAL VARIANTS OF LP

- Bullous lichen planus
- Guttate LP: good prognosis
- Inverse LP
- Hypertrophic lichen planus: severely pruritic in the extremities with hypertrophy.

CLINICAL VARIANTS OF LP

- Follicular LP.

CLINICAL PICTURE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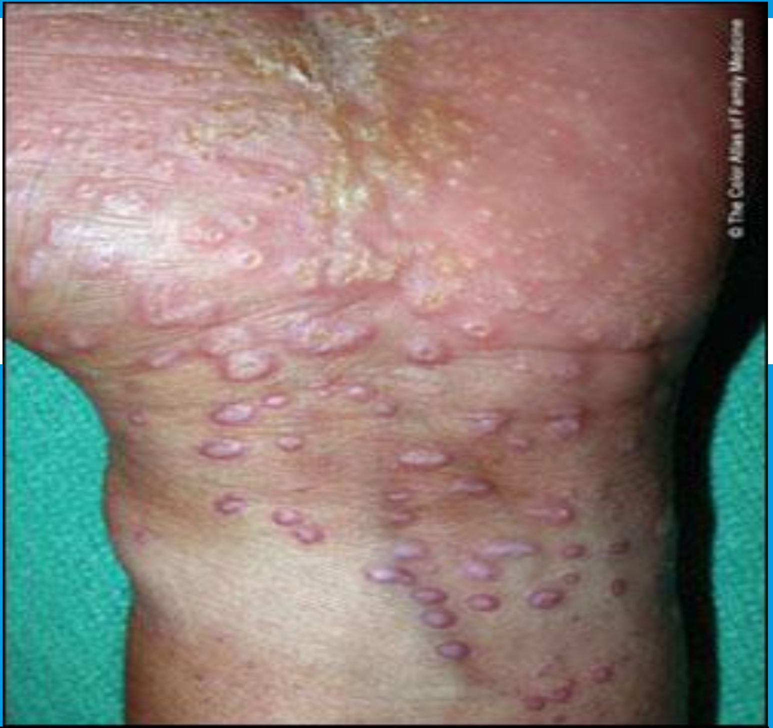
- Papule
- plaque
- Purple
- Polygonal
- Plane
- Pruritic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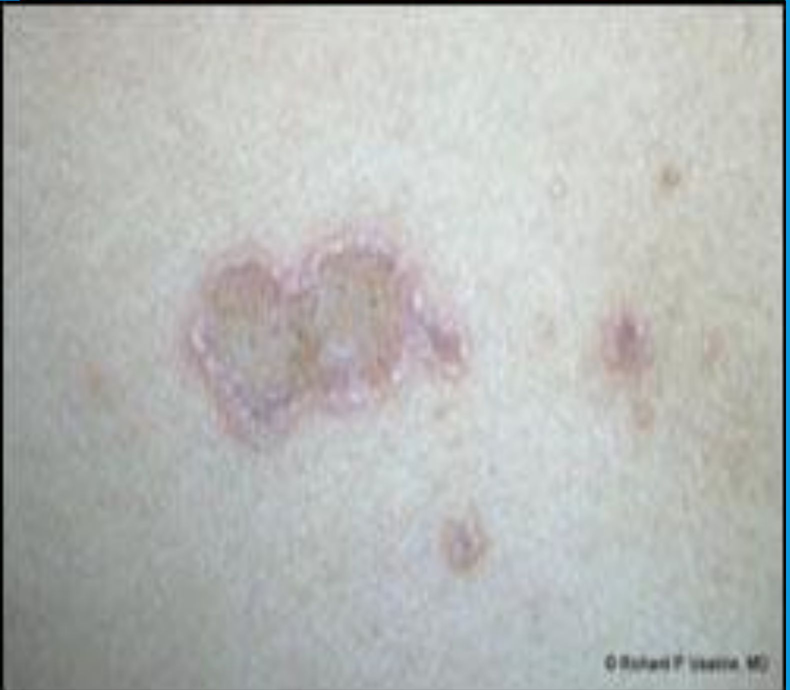
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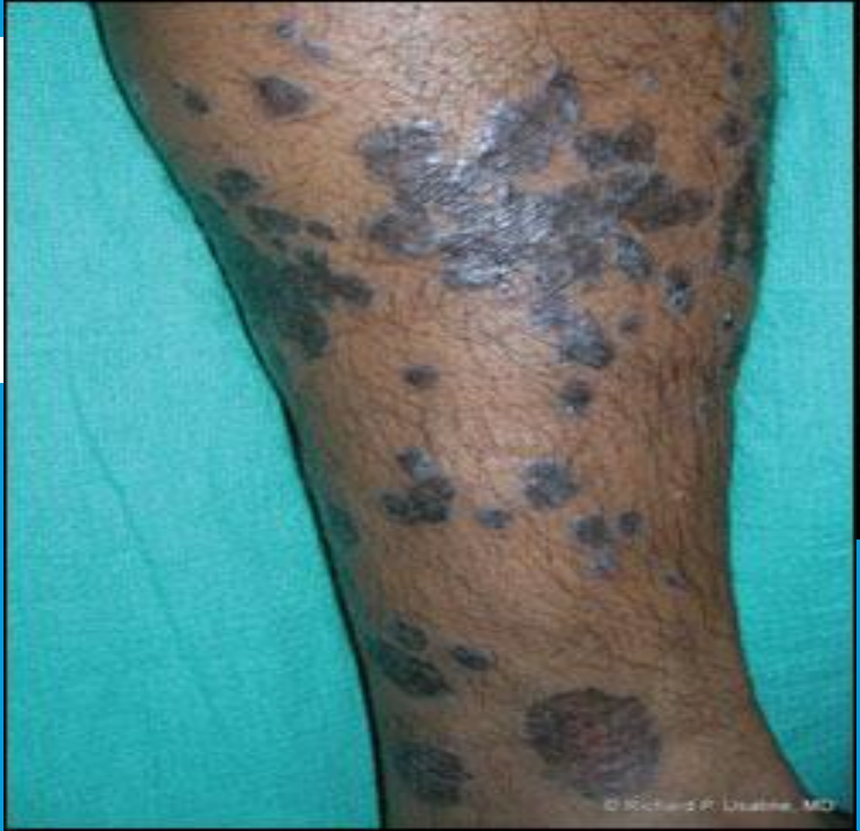
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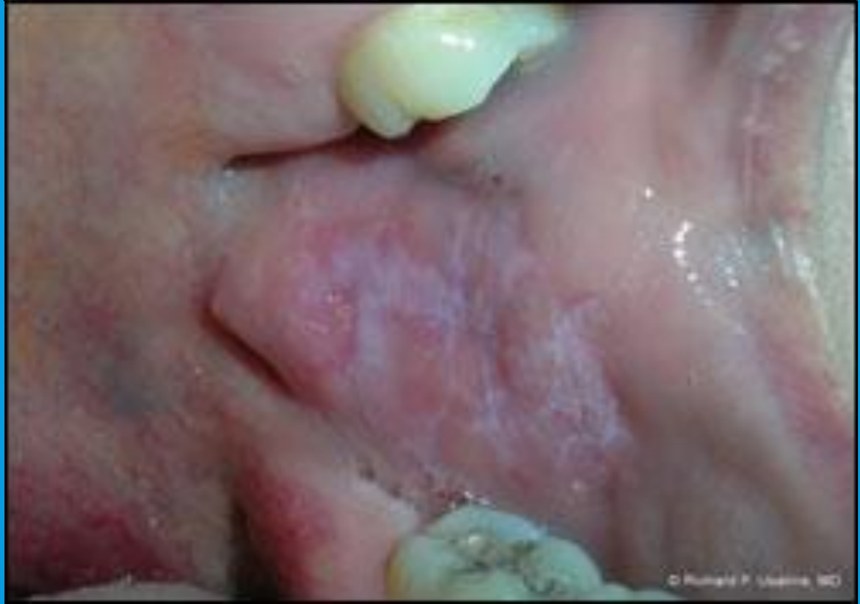
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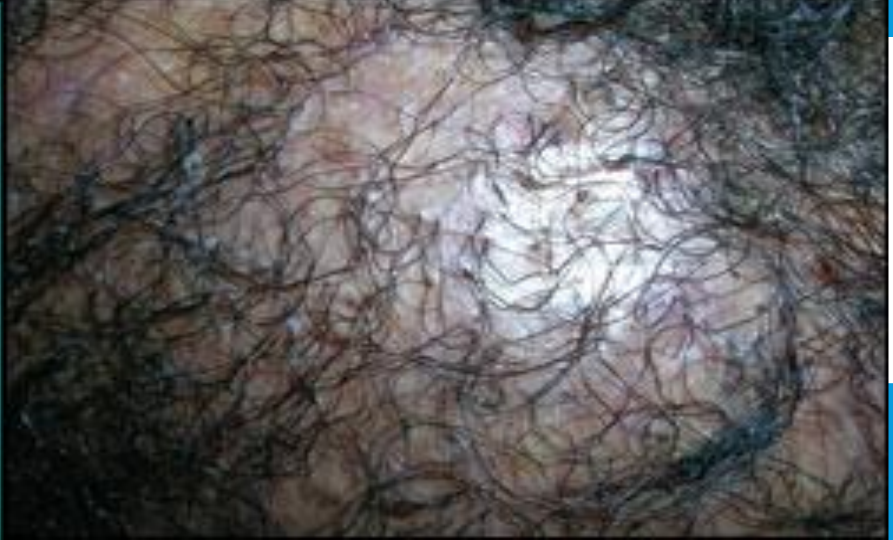

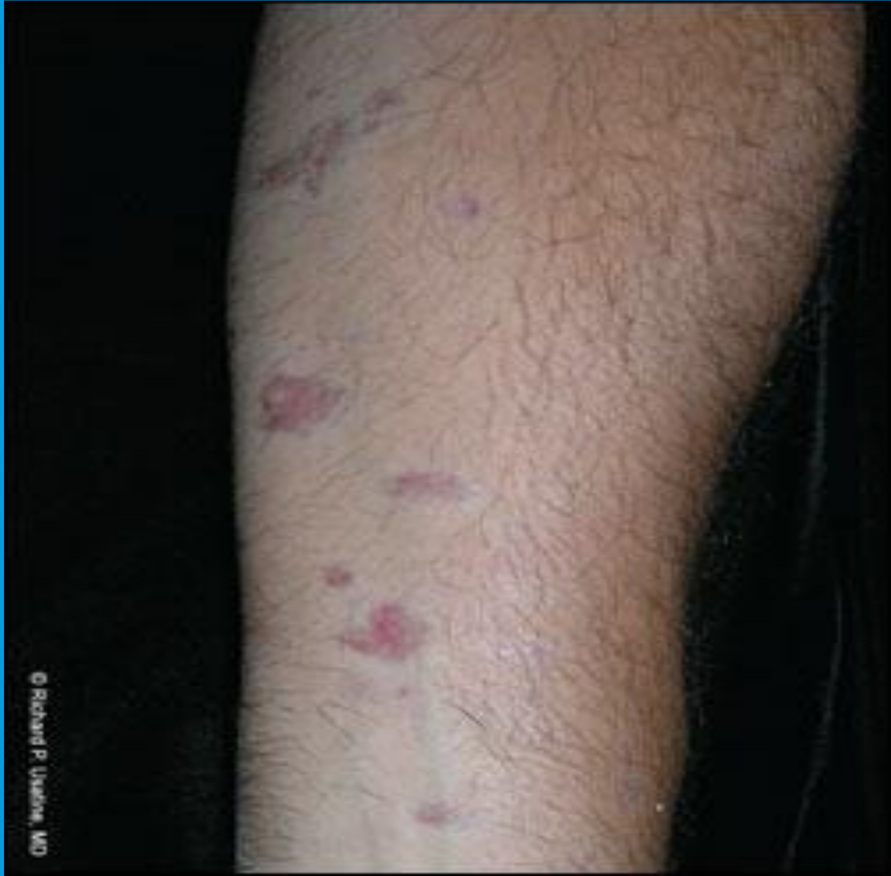
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MANAGEMENT

- High-potency topical corticosteroids
- Oral corticosteroids (prednisone)
- Phototherapy
- *Acitretin*
- Topical calcineurin inhibitors.
- Antihistamines and tranquilizers

PITYRIASIS RUBRA PILARIS

Disorder of keratinization involving children and adults

Clinically there are 5 subtypes where the classical types are characterized by:

1- Follicular hyperkeratosis on erythematous base(first on Knees, elbows)

2- Waxy yellowish keratoderma of the palms and soles.

PITYRIASIS RUBRA PILARIS

3- Scaly scalp

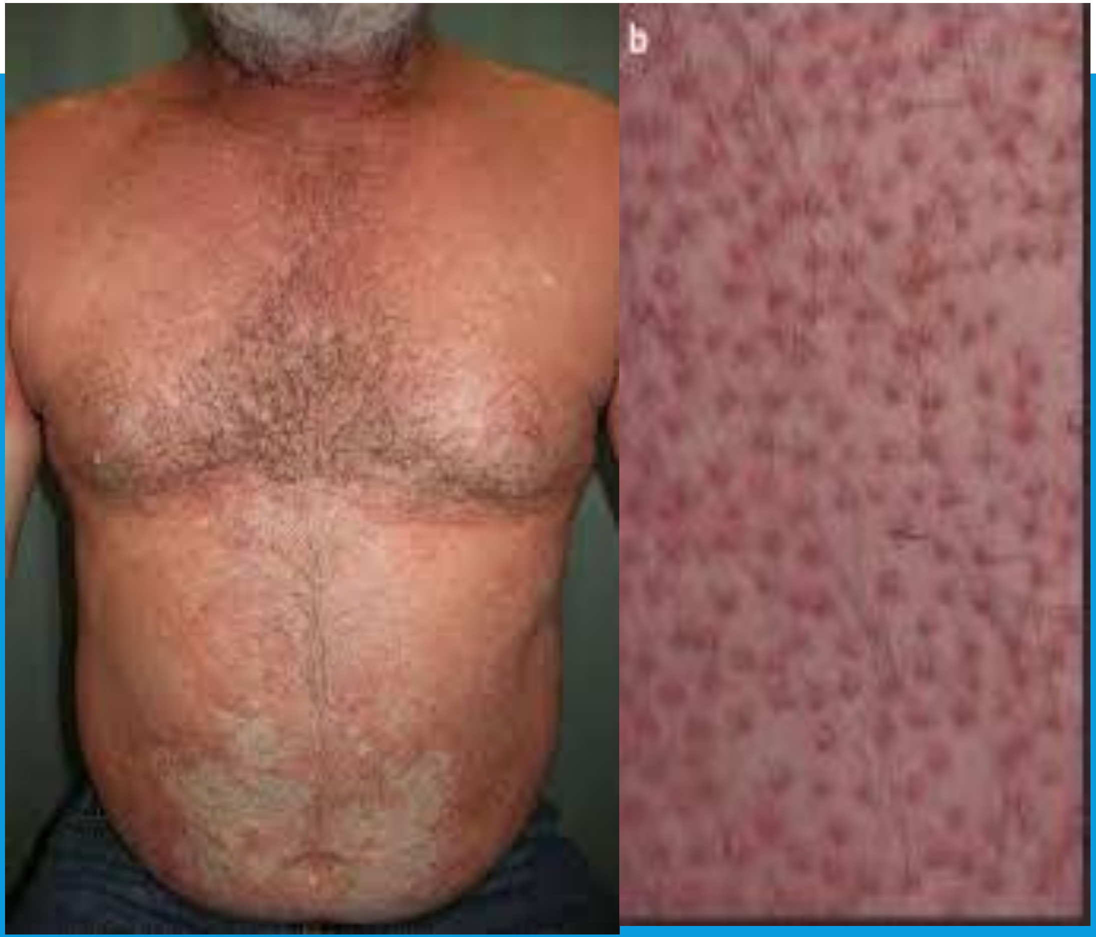
4- Scaly erythema of the face and neck

5- Erythroderma may develop.

6- Islands of normal skin.

7- Nail changes (thickened, brittle, striated)

PITYRIASIS RUBRA PILARIS



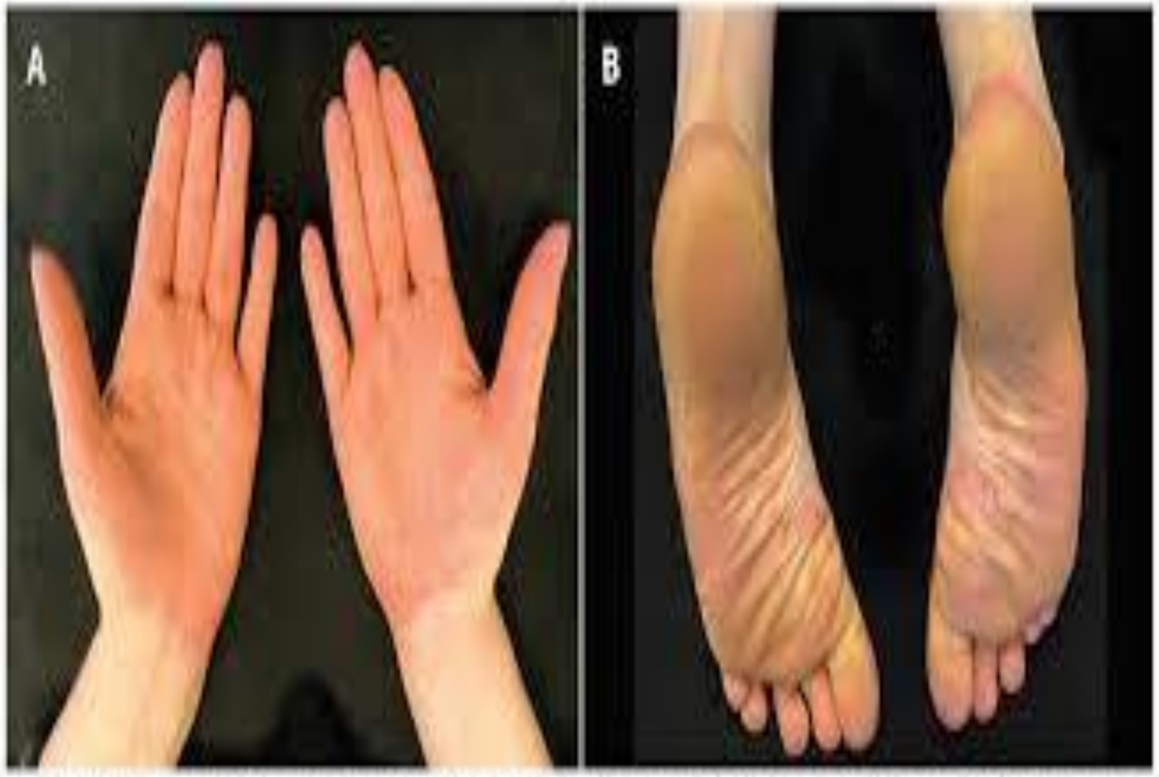


Figure 1: (A) Confluent, red-orange, waxy plaques on the palmar aspects of the hands of a 34-year-old woman with pityriasis rubra pilaris. (B) Similar red-orange plaques on the plantar aspects of the feet.

TREATMENT OF PRP

- Topical corticosteroids and salicylic acid.
- Vit A 150,000-300,000 IU /day
- Accutane 1-1,5mg /day
- Methotrexate 10-25 mg /week
- Narrow band UVB
- PUVA or Re-PUVA.

PITYRIASIS ROSEA

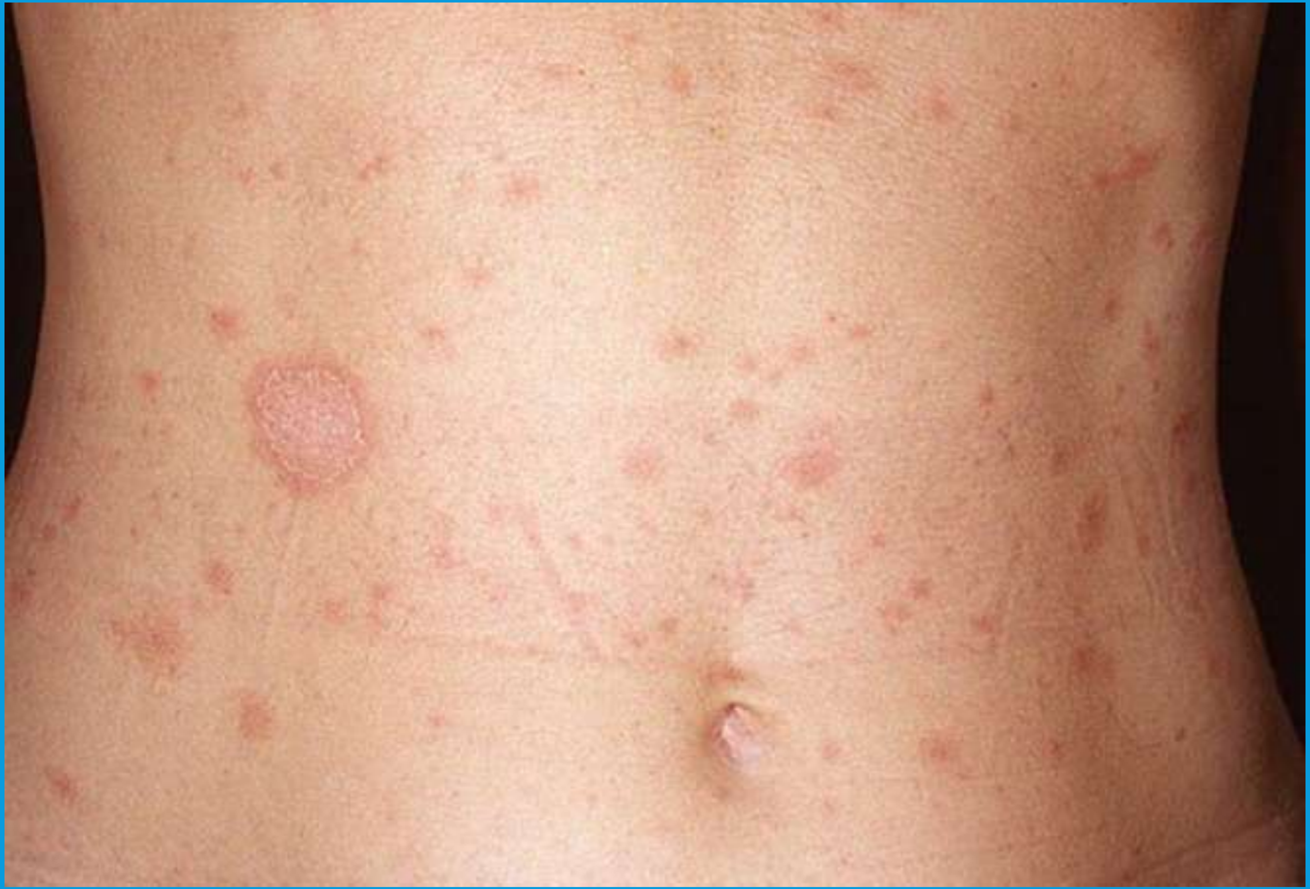
- Self limiting eruption seen mainly in adults
- Caused by HHV6,7
- More common in females and in spring and Autumn.
- Clinically: Herald patch(sharply defined, bright red, covered by collarette of scale at the periphery of the lesion)

PITYRIASIS ROSEA

- Lesions occur in charismas tree pattern, mainly in the trunk, neck, upper third of the arms,

- Fades with in 3-7 weeks.
- C/varieties: inverse, generalized, typical

PITYRIASIS ROSEA



PITYRIASIS ROSEA

Management:

-Re-assurance,

-Emollients ,

-Topical corticosteroids,

-Antihistamines

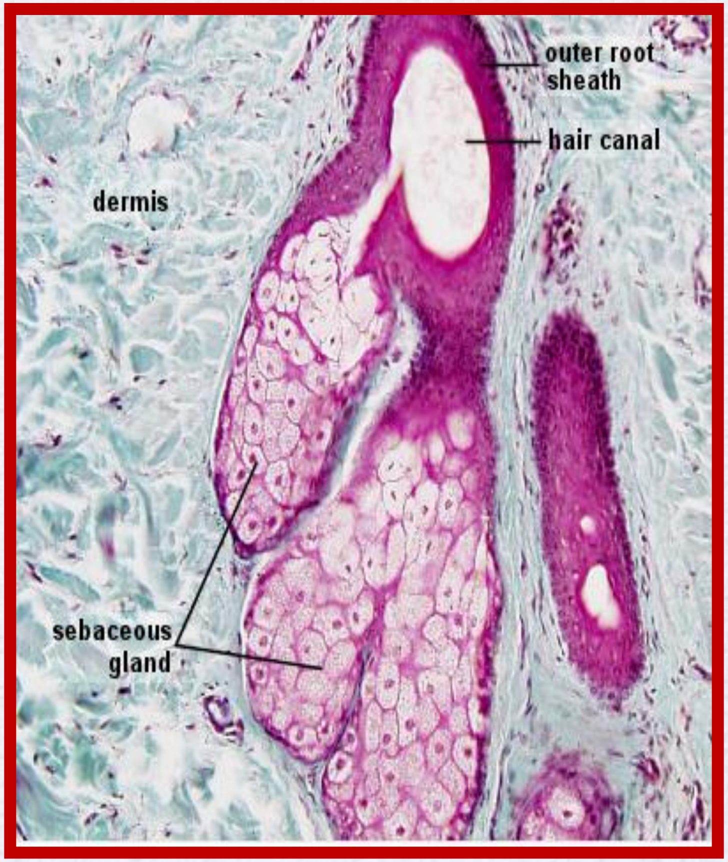
THANK YOU

Sebacous glands disorders

Skin appendages

Sebaceous glands:

- Attached to hair follicles or open freely.
- Present in the scalp, forehead, face,upper chest except palms and soles.
- Secrete sebum to moisturize the skin.
- Sebaceous glands are under the control of androgens.


outer root sheath
hair canal
dermis
sebaceous gland

ACNE:WHY DO WE CARE

- Affects
  - >80% of adolescents
  - >40% of adults over than 25
- Genetics plays a role
- Associated with
  - Disfigurement
  - Pain
  - Loss of confidence
  - Depression
- Effects on quality of life are comparable to those suffering from chronic diseases like asthma, seizures and diabetes

PATHOPHYSIOLOGY

- Typically begins at puberty.
- Disorder of the pilosebaceous unit (face, neck, chest, shoulders, back).
- Increased androgen production leads to increased sebum. Abnormal keratinization and desquamation obstructs the pilosebaceous duct and *Propionibacterium acnes* proliferates in excess sebum and breaks down sebum into free fatty acids. Proinflammatory mediators are activated and result in inflammatory acne.

EXTRINSIC INFLUENCES

- Friction and manipulation
- Occlusive products
- Medications:
  - Steroids
  - Antiepileptics
  - Progestin only contraceptives

DIET, STRESS and ACNE

- Controversial link between diet and acne although many patients believe that their acne is influenced by certain foods
- Western diet may be associated with acne
- Skim milk is associated with acne in teenage girls
- Stress: acne among university students was associated with exam stress

CLINICAL FEATURES

Mild acne

- Comedomes:
  - Closed (whiteheads) are closed flesh colored papules 1-3mm in size
  - Open (blackheads) are open and the contents of the comedome oxidizes upon expose to the light (tyrosine is oxidized to melanin)

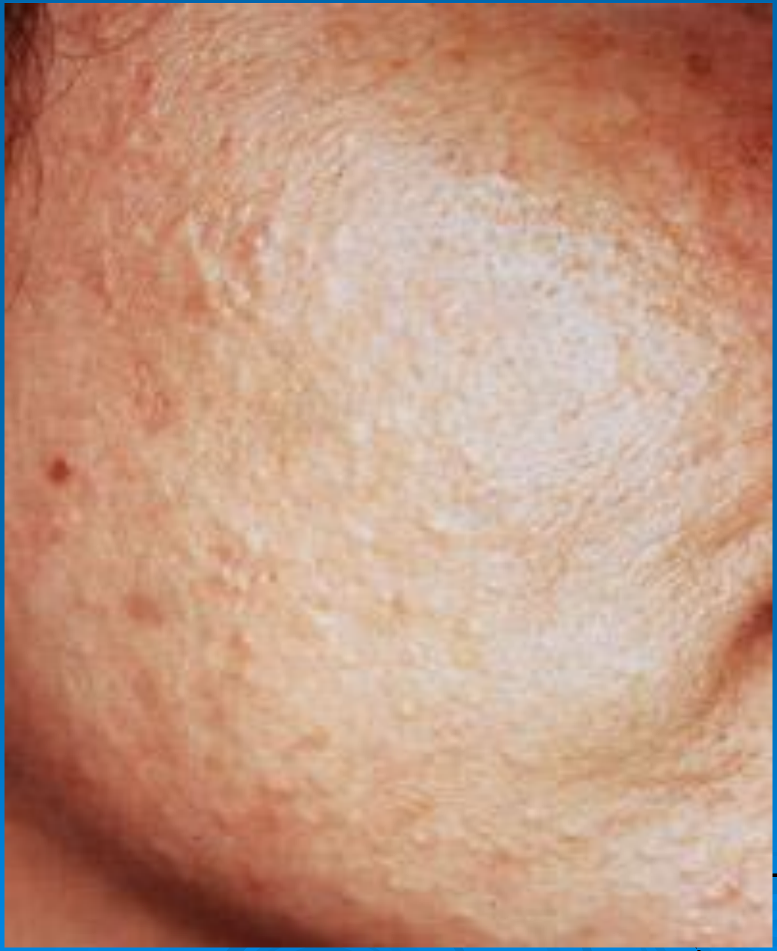
Moderate acne

- Comedomes/Papules/Pustules

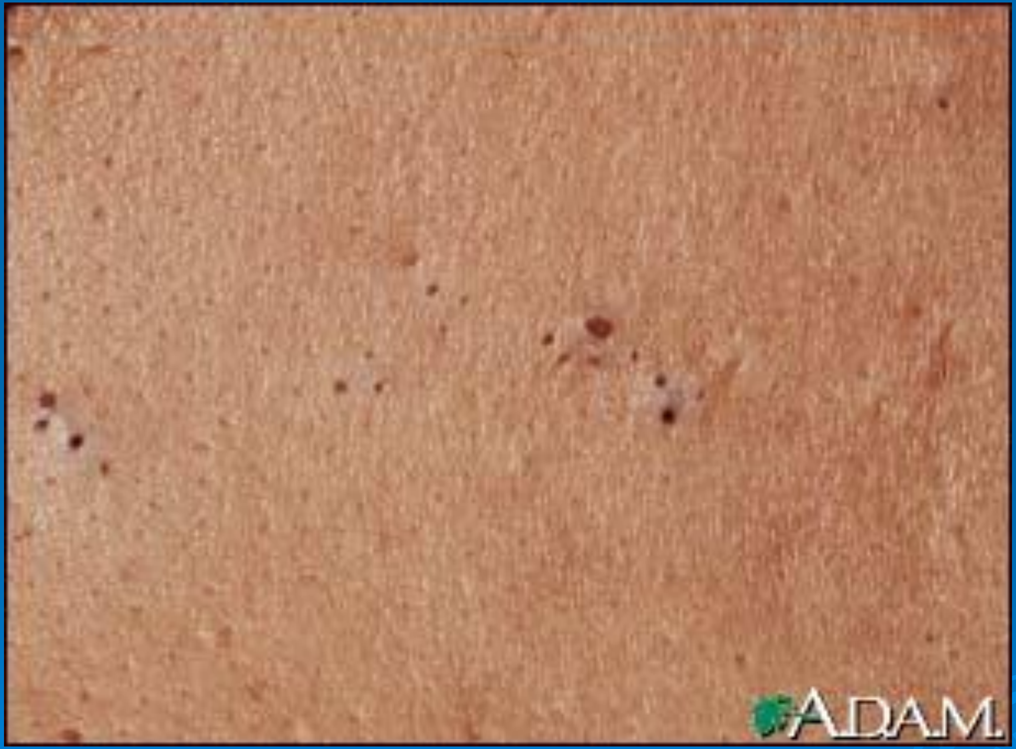
Severe acne

- Papules/Pustules/Nodulocystic lesions

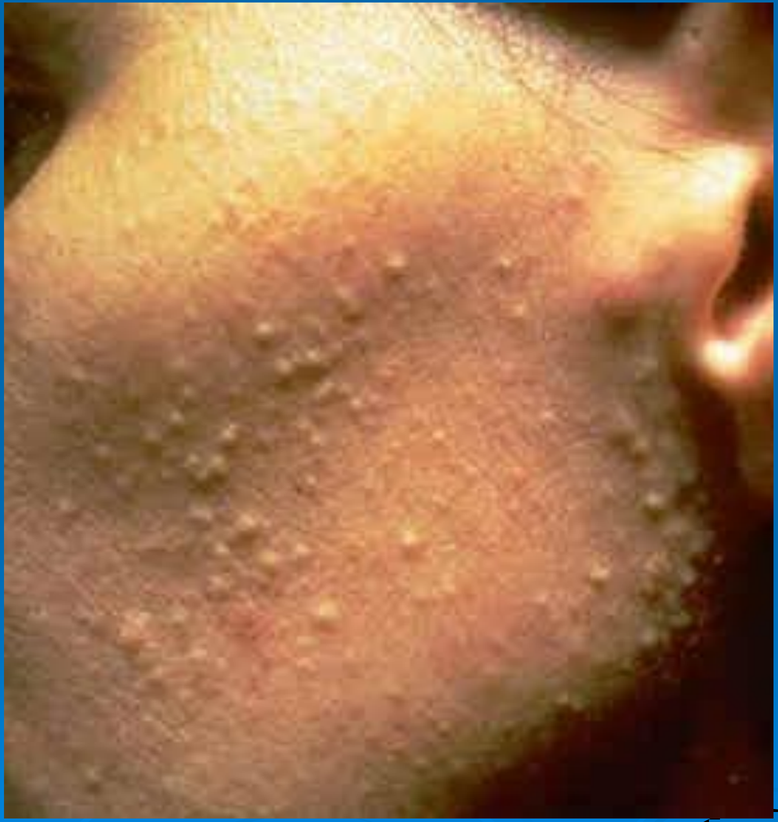
CLOSED COMODOME



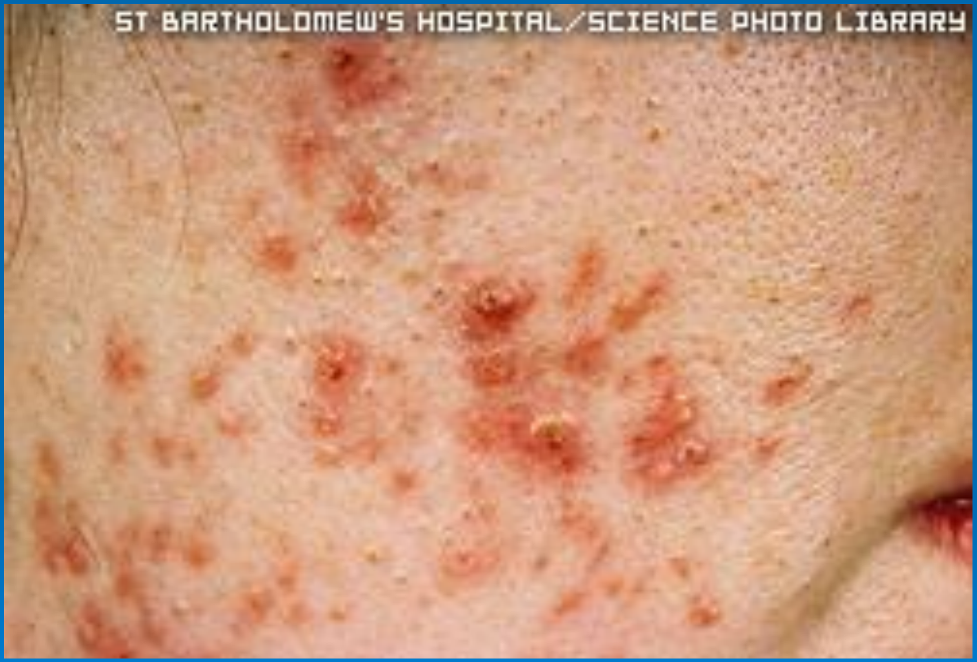
OPEN COMODOME



PAPULAR ACNE



PUSTULAR ACNE



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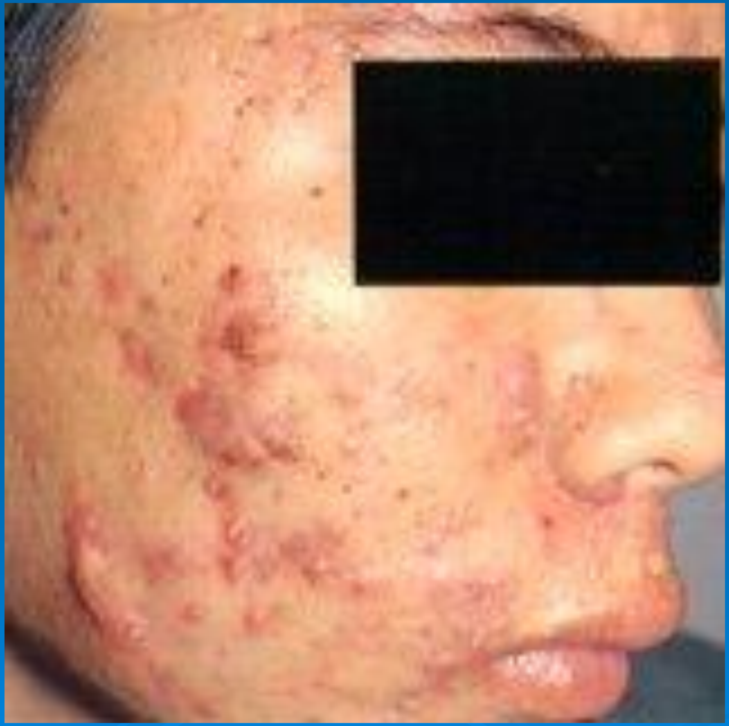
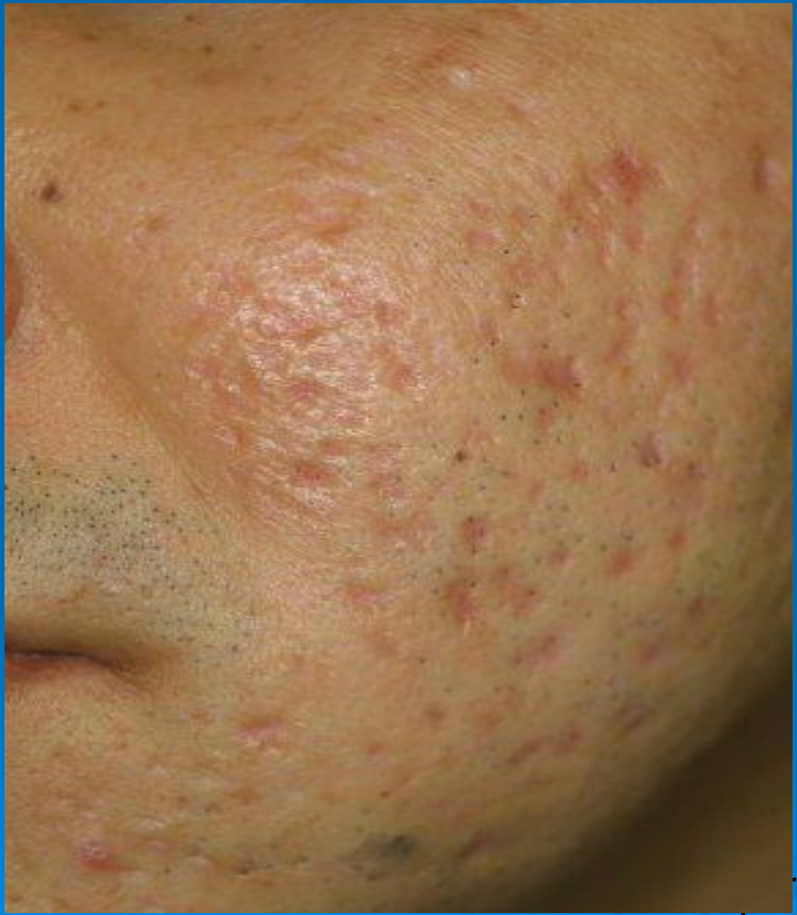
NODULOCYSTIC ACNE



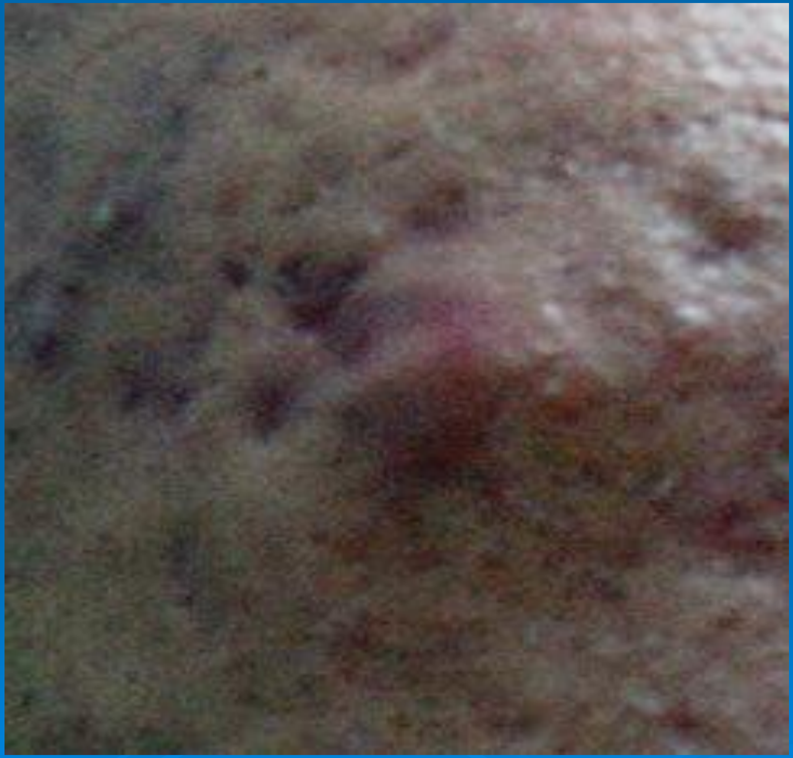
CLINICAL FEATURES

- Postinflammatory changes can occur with healing and resolve over time
- Risk of scarring
  - Mild acne low risk
  - Moderate acne medium risk
  - Severe acne high risk c/o punctate depressions (ice-pick scars), depressed scars (thumbprint scars), hypertrophic papular scars, keloids

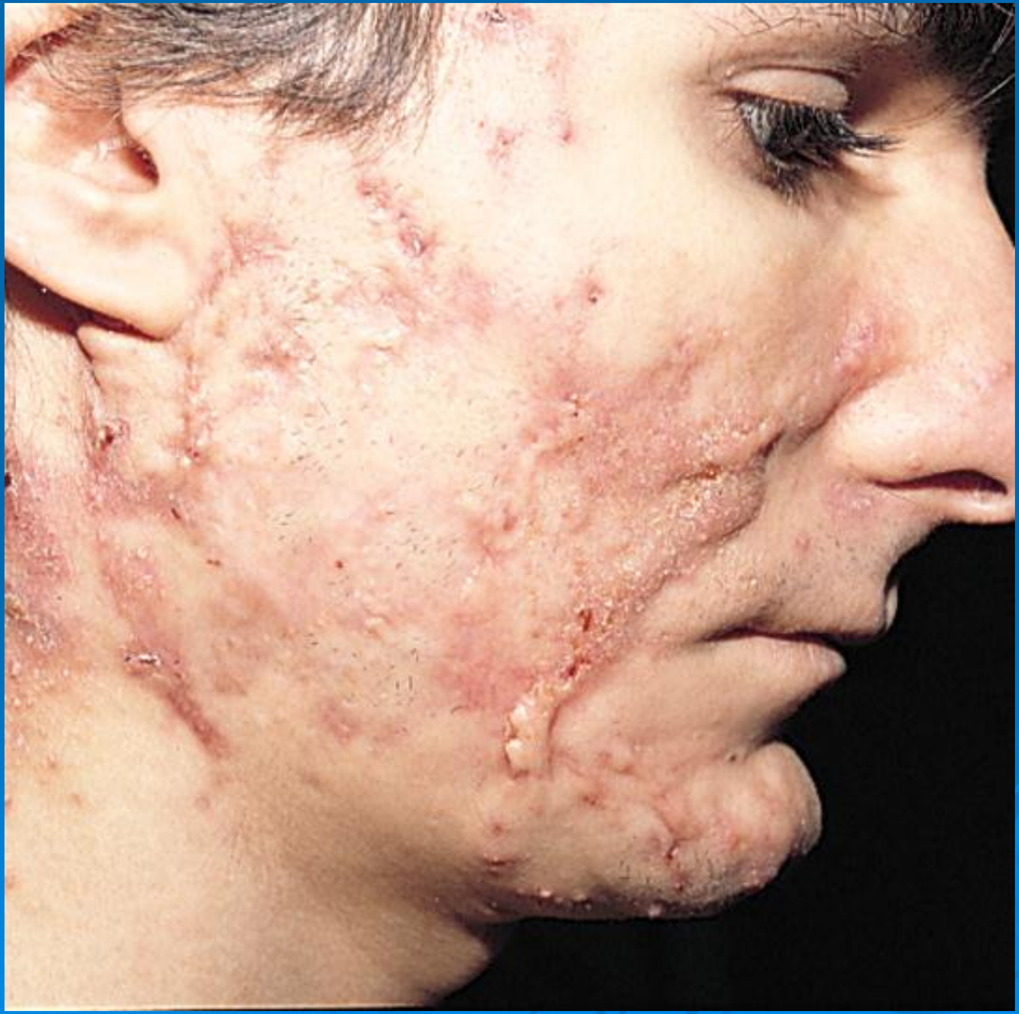
ICE PICK SCAR



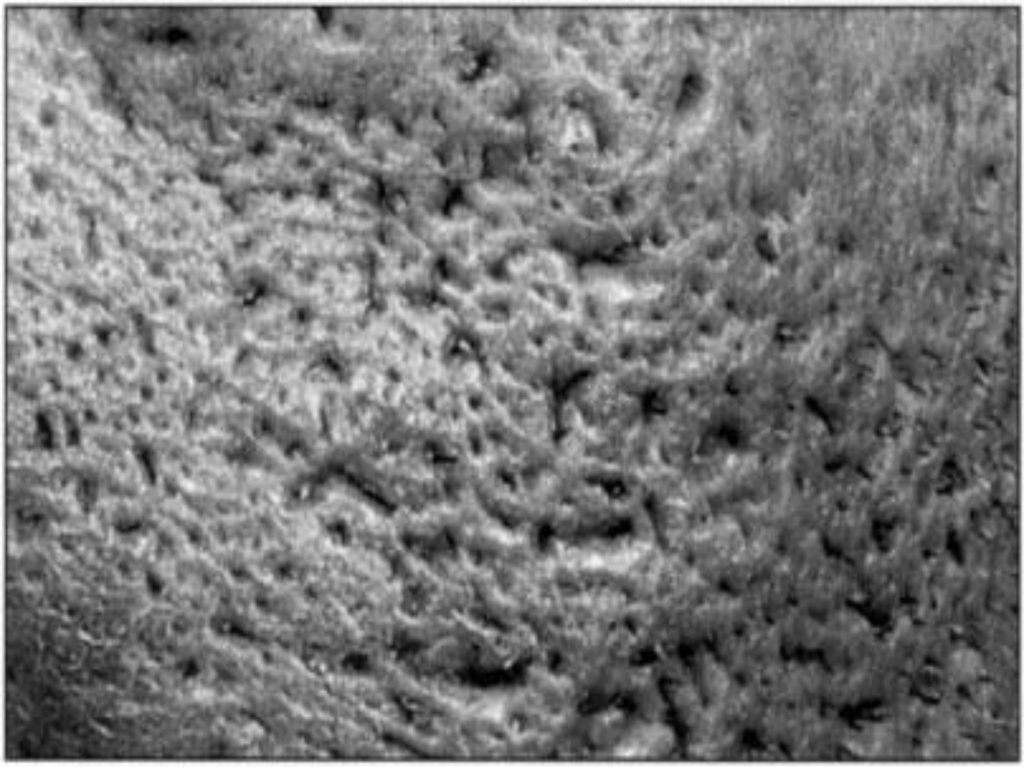
THUMBPRINT SCARS



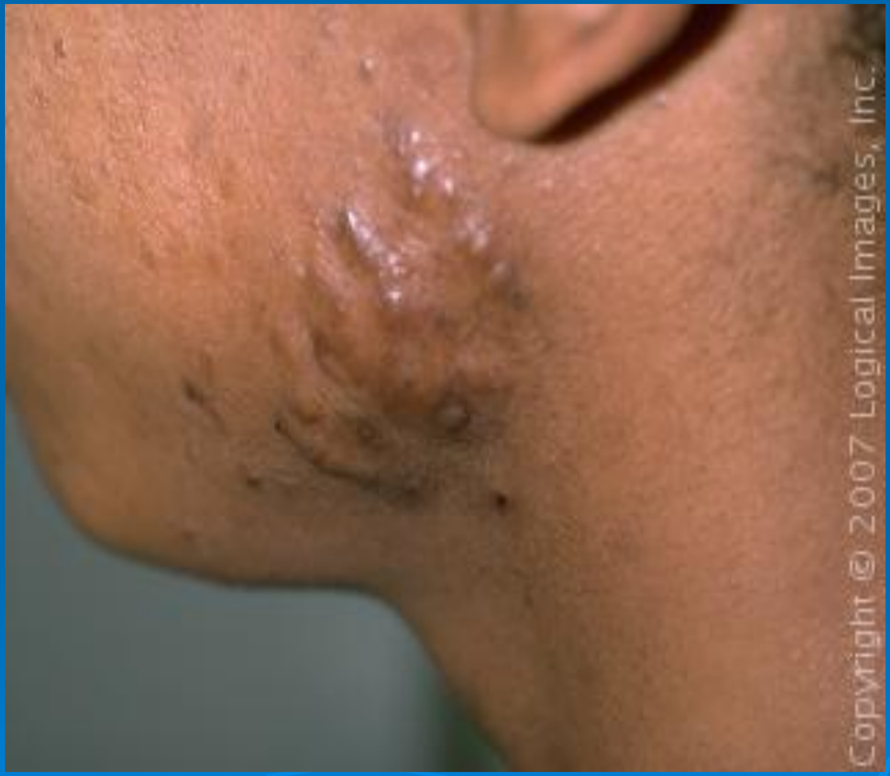
ATROPHIC SCARS



HYPERTROPHIC SCARS



KELOIDS



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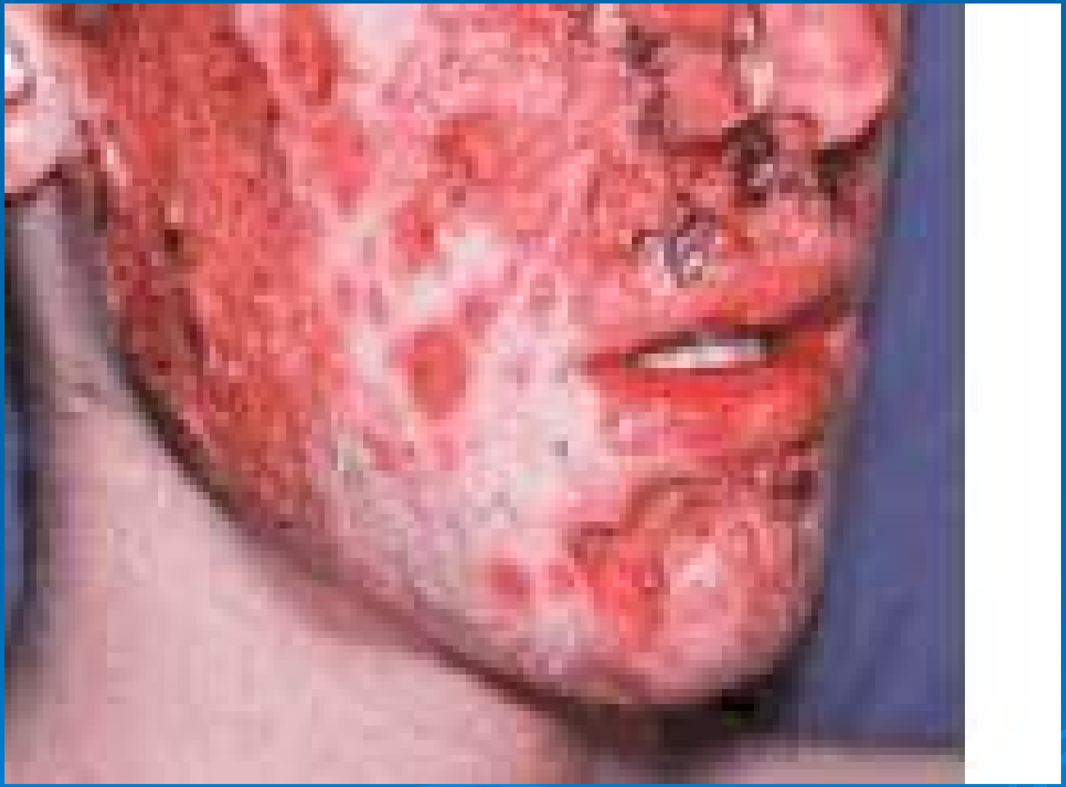
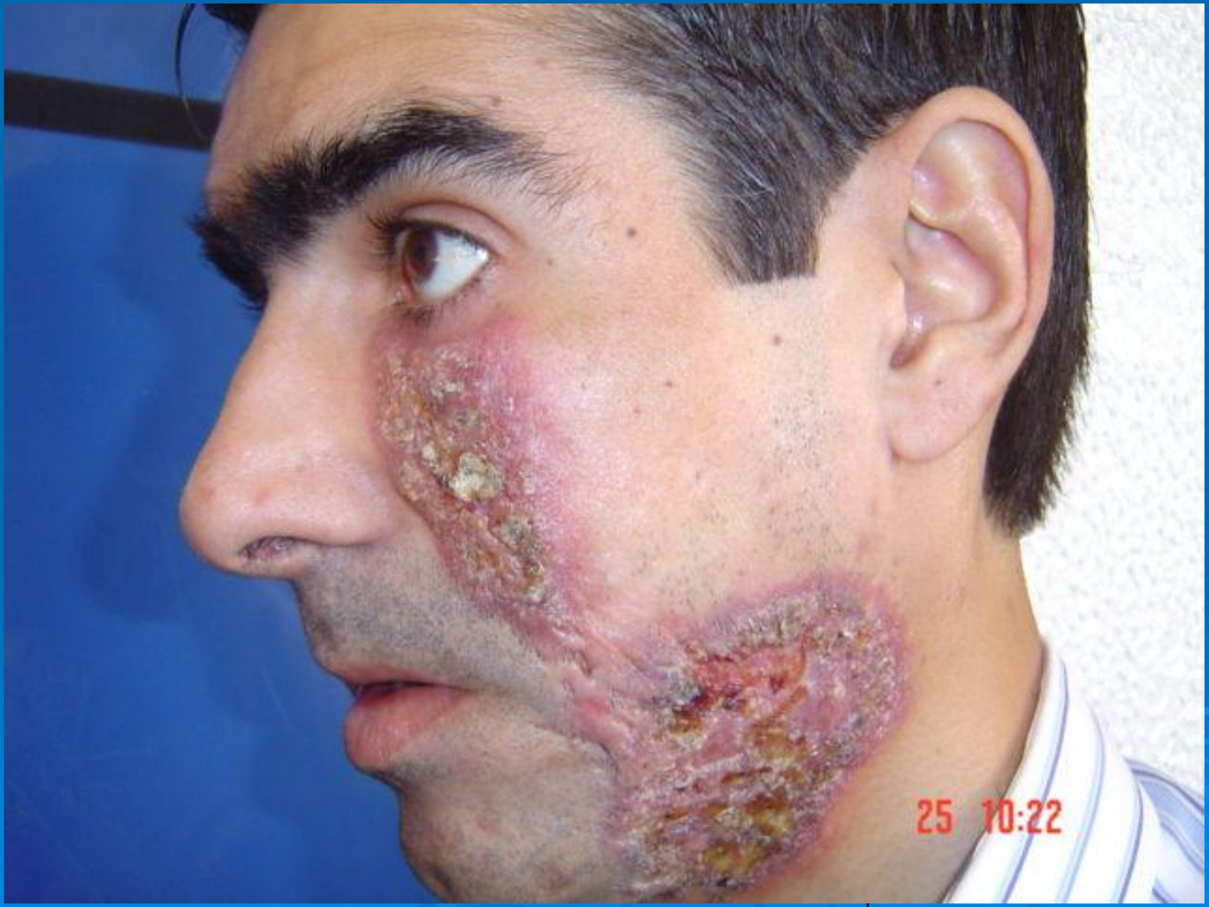
SEVERE ACNE

- Acne fulminans
  - Severe acne in young males in association with fever, arthritis
- Acne conglobata
  - Comedomes, pustules, foul smelling cysts, sinus tracts, atrophic and keloid scarring
- Treat with high dose steroids

ACNE FULMINANS



ACNE CONGLOBATA



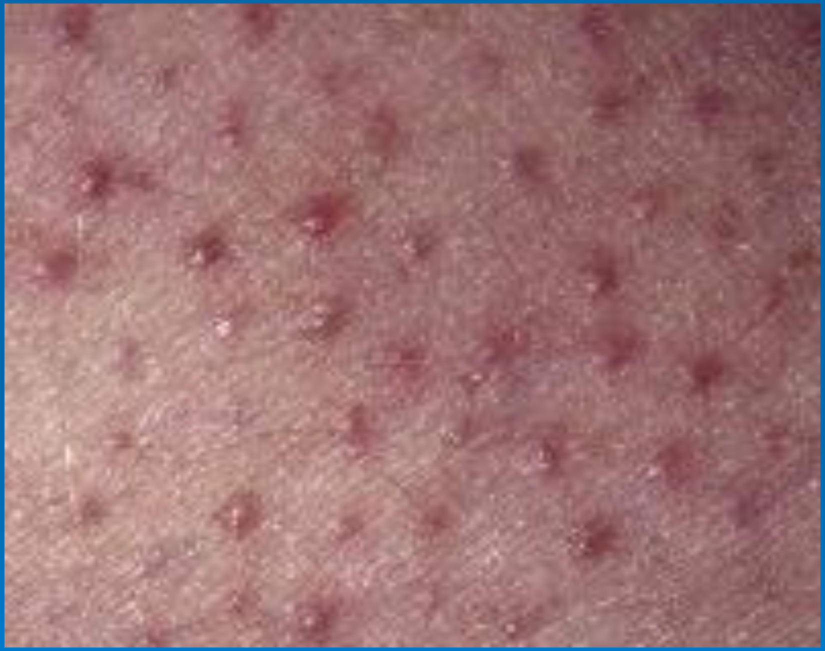
DIFFERENTIAL DIAGNOSIS

- Keratosis pilaris
- Perioral dermatitis
- Angiofibromas
- Pseudofolliculitis barbae
- Acne keloidalis nuchae
- Folliculitis
- Hidradentis suppurativa

KERATOSIS PILARIS

- Small perifollicular papules on the face and the extensor surfaces of the arms and legs
- May be seasonal
- May improve w/ keratolytic moisturizers containing ammonium lactate or urea

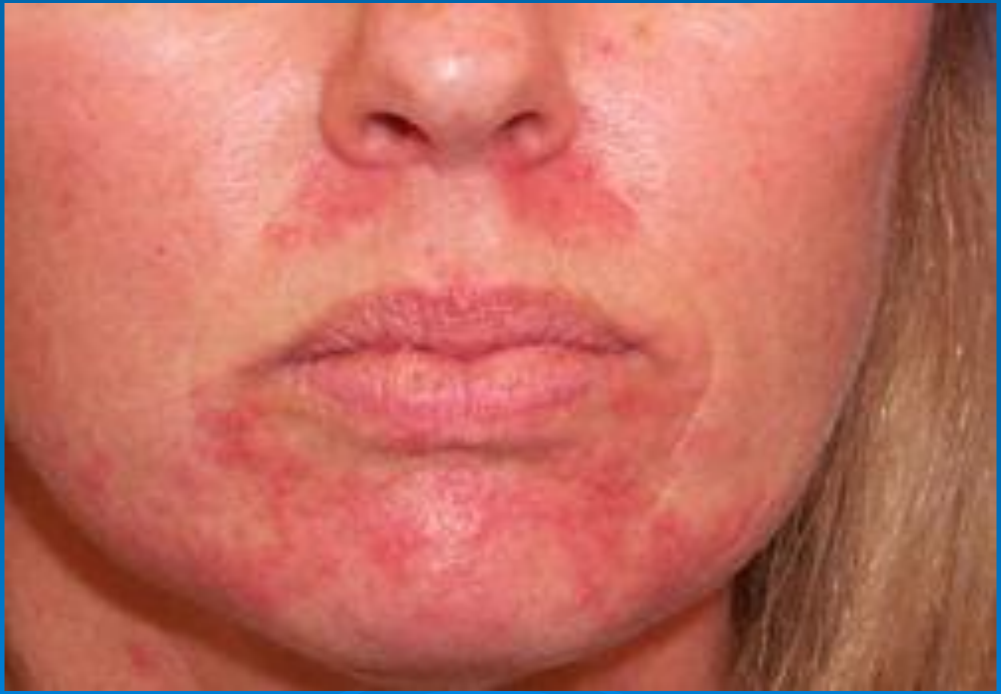
KERATOSIS PILARIS



PERIORAL DERMATITIS

- Idiopathic
- May occur after use of topical steroids
- Treatment- Discontinue steroid use, topical benzoyl peroxide, topical antibiotics

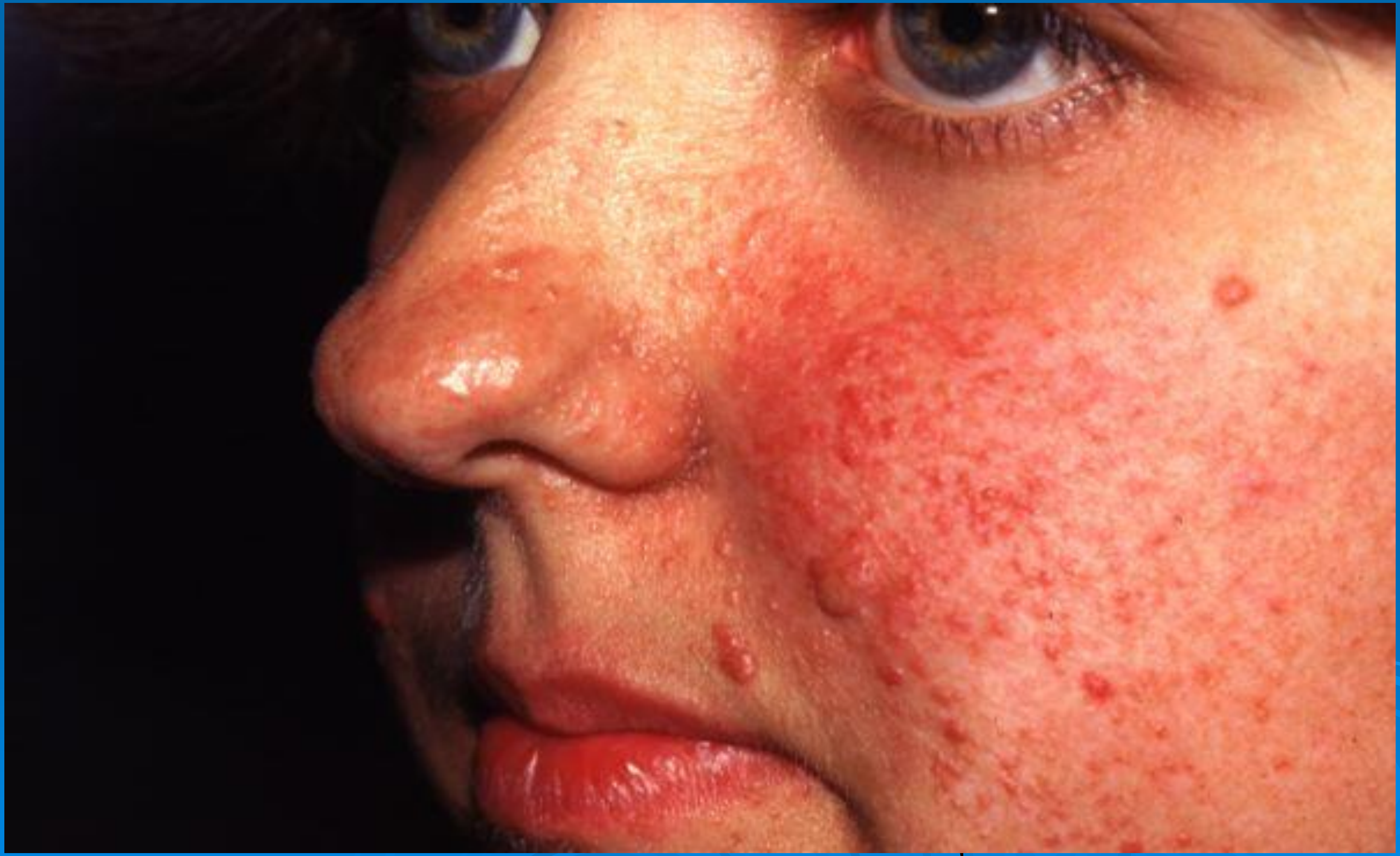
PERIORAL DERMATITIS



ANGIOFIBROMAS

- Tuberous sclerosis
- Rubbery papules/plaques
- Flesh colored to brownish
- Seen on nasolabial folds
- Begin in childhood
- Treat with pulsed dye laser therapy or carbon dioxide laser resurfacing

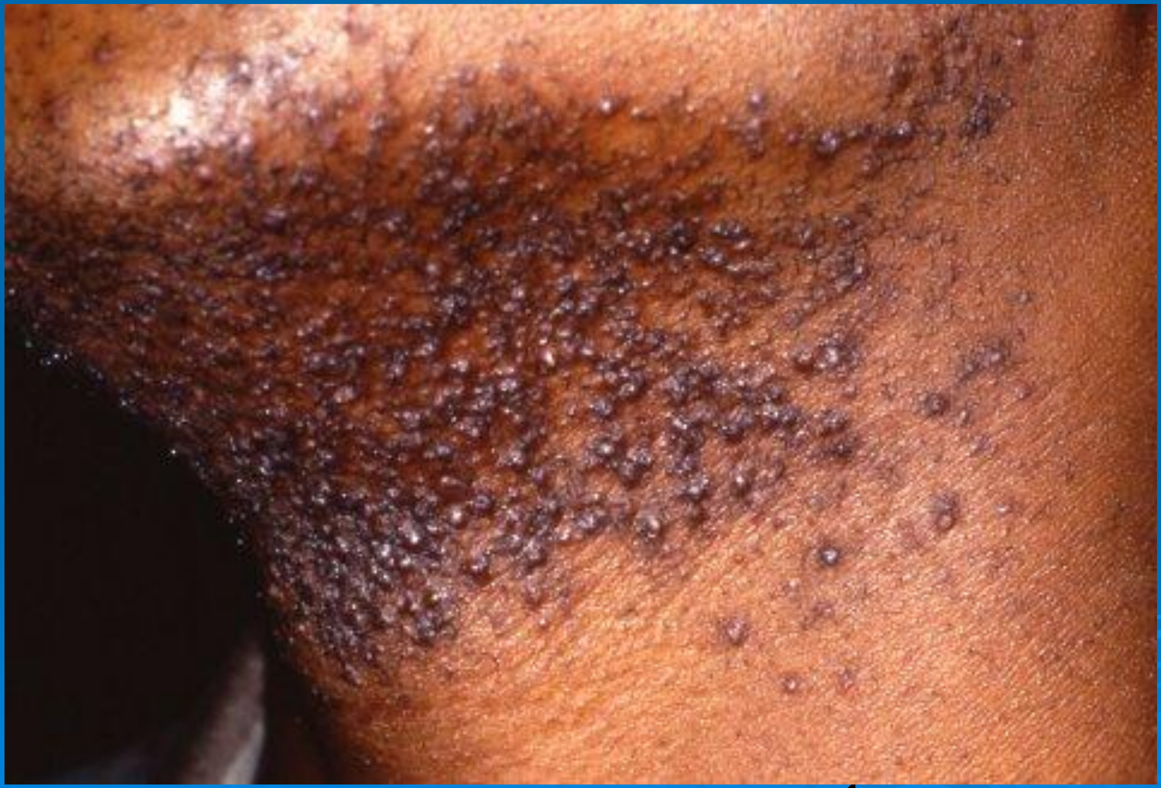
ANGIOFIBROMAS



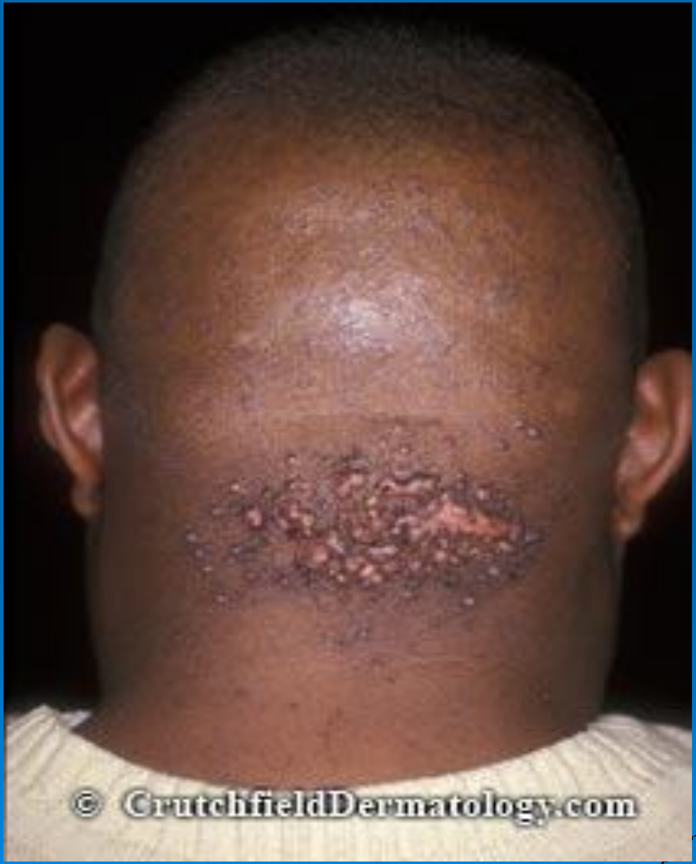
SHAVING

- Pseudofolliculitis barbae
  - Beard hair, when shaved closely, causes inflammation, papules and nodules
- Acne keloidalis nuchae
  - Papules and nodules on the nape of the neck
- Avoid close shaves, use depilatories, topical retinoids, benzoyl peroxide

PSEUDOFOLLICULITIS BARBAE



ACNE KELOIDALIS NUCHAE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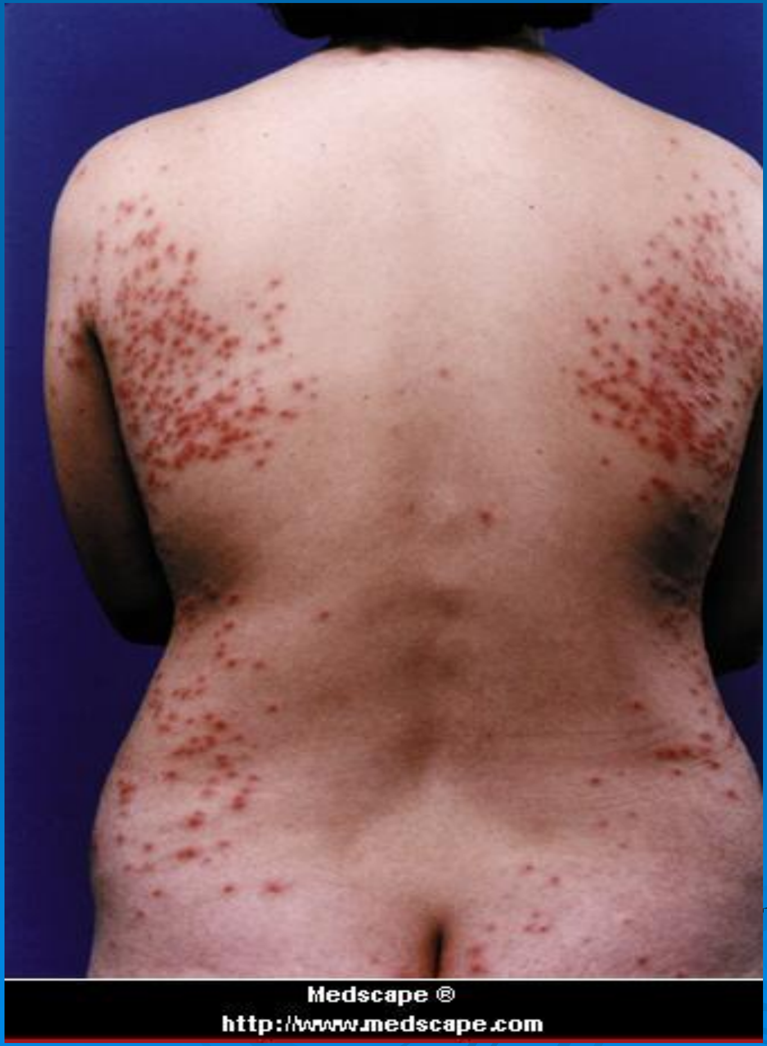
FOLLICULITIS

- Papules/pustules on the face, back buttocks
- Typically *staph aureus*
- Benzoyl peroxide or topical antibiotics may help prevent outbreaks

FOLLICULITIS



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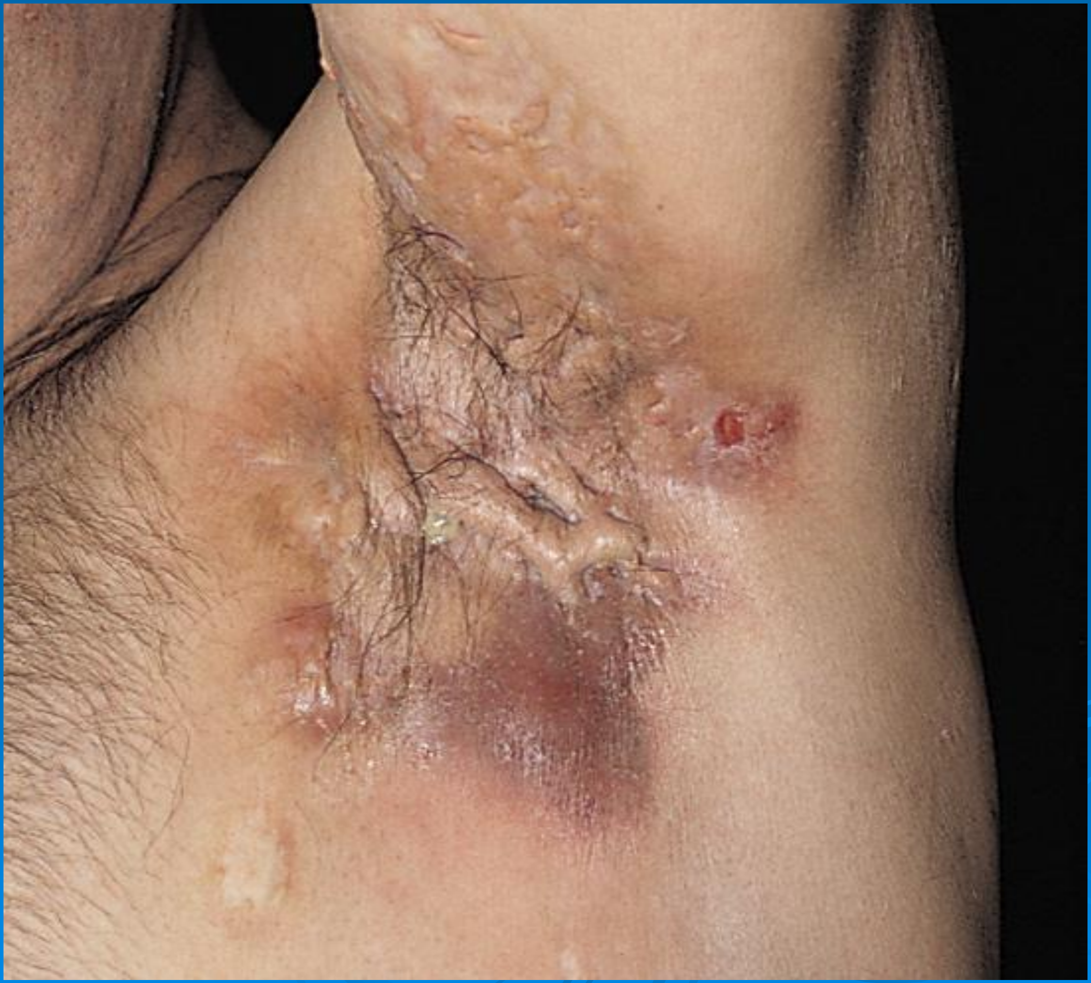
HIDRADENITIS SUPPURATIVA

- Disease of the follicle
- Deep tender nodules in the groin, axilla, buttocks
- Difficult to treat
- May respond to Accutane

HIDRADENITIS SUPPURATIVA



TOPICAL RETINOIDS

- Tretinoin (Retin A), Adapalene (Differin), Tazarotene (Tazorac)
  - Excellent choice for comedomal acne
  - Improves follicular desquamation and dyschromia
  - Anti-inflammatory action (Differin best)
  - Use at night over entire face, exposure to the sun increases irritation
  - Results in six to eight weeks. May increase concentration over time
  - Degraded by prolonged exposure to the sun and when used with benzoyl peroxide (Differin most photostable)

TOPICAL RETINOIDS

- Adverse affects
  - Irritant potential (Tazorac most irritating, Differin least)
  - Sun sensitivity
  - Pustular eruption after 3-4 weeks
  - Potential hyper/hypopigmentation in black and Asian patients
  - Contraindicated in pregnancy

TOPICAL ANTIBIOTICS

- Erythromycin, Clindamycin,
  - Decrease *P.acnes* and percentage of free fatty acids
  - Slow to act
  - Resistance often develops over time
  - Best used in combination with topical retinoids/benzoyl peroxide
  - Rare cases of pseudomembranous colitis w/ topical clindamycin

BENZOYL PEROXIDE

- Bactericidal effect on *P.acnes*
- No evidence of resistance
- BP combined with a topical antibiotic may help decrease the presence of antibiotic resistant *P. acnes*
- Mild comedolytic action, decreases free fatty acids
- Adverse effects
  - Irritation
  - Bleaches clothing and hair
  - Allergic contact dermatitis

AZELAIC ACID

- Dicarboxylic acid that is bacteriostatic against *P.acnes* and normalizes keratinization
- Most effective when used with other agents
- Side affects uncommon
- Use in caution in teens w/ dark complexions due to potential risk of hypopigmentation

SYSTEMIC ANTIBIOTICS

- Primarily used for moderate to severe inflammatory acne
- Decreases *P.acnes*
- Reduces amount of free fatty acids
- Preferred agents: Tetracyclin, Doxycyclin, Minocyclin
- High rates of resistance to Erythromycin

HORMONAL CONTROL

- Oral contraceptive pills in females
  - Increases production of sex hormone binding globulin leading to a decrease of circulating androgens
  - Decreases ovarian androgen production
- Ortho tri-cyclen, Estrostep FDA approved for the treatment of acne
- Oral antiandrogens (spironolactone) can be useful
- Oral corticosteroids, short course for patients with severe inflammatory disease

ISOTRETINOIN

- Systemic retinoid used for nodulo-cystic acne
- Most effective treatment with remission in 60% after single course (15-24 weeks)
- Reduces sebum production
- Normalizes follicular keratinization
- Decreases inflammation
- Baseline CBC, LFT's, cholesterol, triglycerides, urinalysis, pregnancy test. Repeat monthly
- Post pubertal females must be on contraception and have two sequential negative pregnancy tests before starting

ISOTRETINOIN

- Adverse effects
  - Teratogenic (facial dysmorphism, abnormalities of brain, eye, ear, CV system, thymus) and retinoid embryopathy can occur with single exposure during gestation
  - Drying/chapping of skin and mucous membranes
  - Myalgias/arthralgias
  - Photosensitivity
  - GI effect: transaminitis, lipid abnormalities, pancreatitis
  - Hematological: leucopenia, elevated platelets and ESR
  - Neurological: pseudo tumor cerebri
  - Renal: proteinuria, hematuria
  - Mood disorders, depression, suicidal ideations and suicides

MISCELLANEOUS THERAPY

- Comedome removal
  - May be helpful if comedomes are resistant to other treatments
- Chemical peels
  - Little evidence supporting efficacy
- Intralesional steroids
  - Used for large inflammatory nodules/cysts
  - Can be associated with local atrophy

OVERVIEW OF THERAPY

- Mild acne: Topical therapy with retinoid for comedomes, add BP or topical antibiotic if mild inflammation present
- Moderate acne: Topical therapy plus oral antibiotics for inflammatory lesions, add BP to reduce antibiotic resistance. Consider OCP's
- Severe acne: Accutane if topical therapy and oral antibiotics fail

URTICARIA

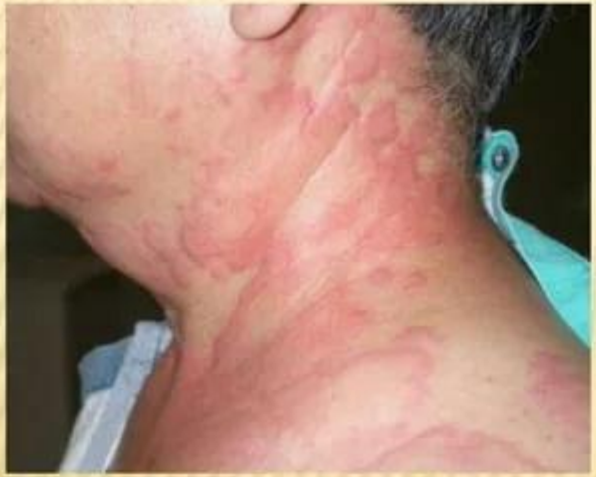
URTICARIA: THE BASICS

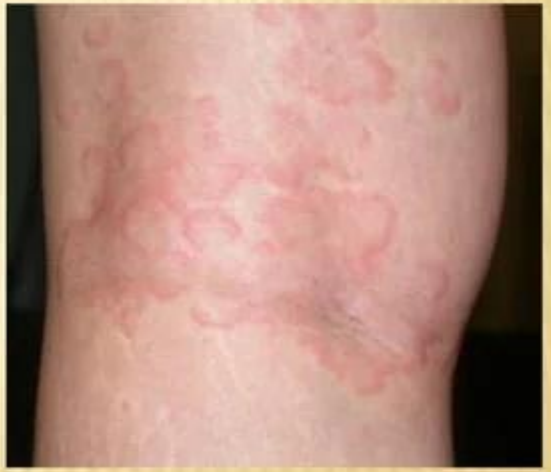
- Urticaria (hives) is a vascular reaction of the skin characterized by wheals surrounded by a red halo or flare (area of erythema)
- Cardinal symptom is PRURITUS (itch)
- Urticaria is caused by swelling of the upper dermis
- Up to 20% of the population experience urticaria at some point in their lives

ANGIOEDEMA: THE BASICS

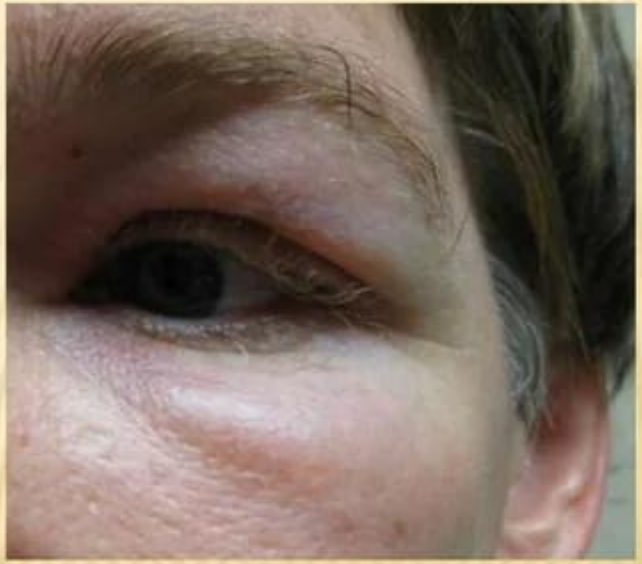
- Angioedema can be caused by the same pathogenic mechanisms as urticaria, but the pathology is in the deep dermis and subcutaneous tissue and swelling is the major manifestation
- Angioedema commonly affects the face or a portion of an extremity
  - Involvement of the lips, cheeks, and periorbital areas is common, but angioedema also may affect the tongue, pharynx, larynx and bowels
- May be painful or burning, but not pruritic
- May last several days

EXAMPLES OF URTICARIA





EXAMPLE OF ANGIOEDEMA



URTICARIA & ANGIOEDEMA

- Urticaria and angioedema may occur in any location together or individually.
- Angioedema and/or urticaria may be the cutaneous presentation of anaphylaxis, so assessment of the respiratory and cardiovascular systems is vital!

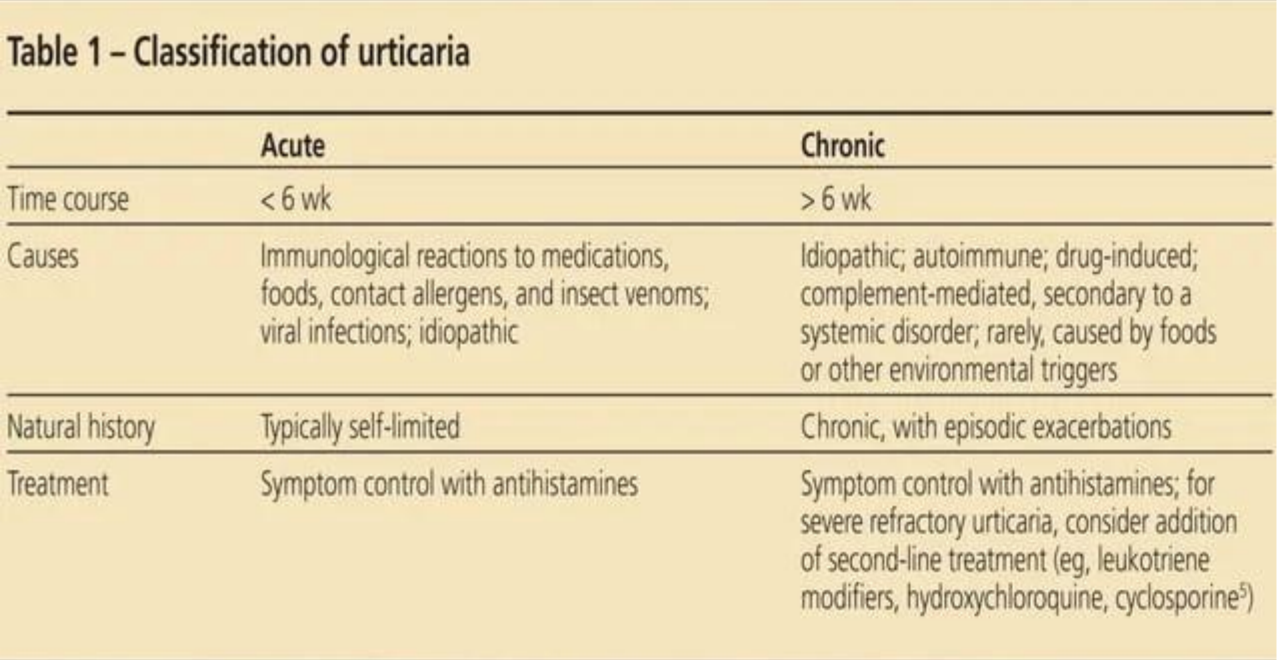


Table 1 – Classification of urticaria

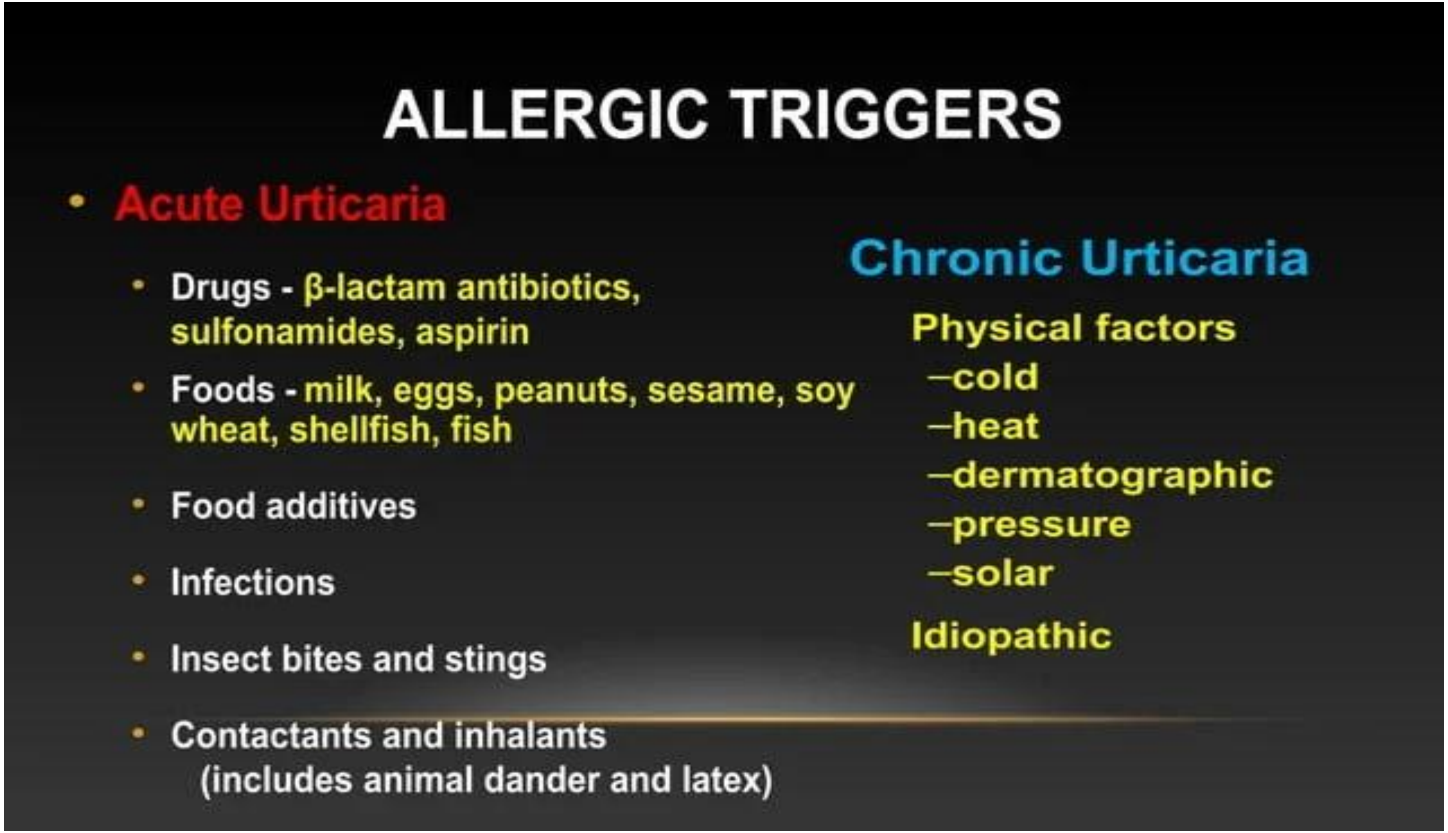
	Acute	Chronic
Time course	< 6 wk	> 6 wk
Causes	Immunological reactions to medications, foods, contact allergens, and insect venoms; viral infections; idiopathic	Idiopathic; autoimmune; drug-induced; complement-mediated, secondary to a systemic disorder; rarely, caused by foods or other environmental triggers
Natural history	Typically self-limited	Chronic, with episodic exacerbations
Treatment	Symptom control with antihistamines	Symptom control with antihistamines; for severe refractory urticaria, consider addition of second-line treatment (eg, leukotriene modifiers, hydroxychloroquine, cyclosporine[5])

URTICARIA: CLINICAL FINDINGS

- Lesions typically appear over the course of minutes, enlarge, and then disappear within hours
- Individual wheals rarely last >12hrs
- Surrounding erythema will blanch with pressure
- Urticaria may be acute or chronic
  - **Acute** = new onset urticaria < 6 weeks
  - **Chronic** = recurrent urticaria (most days) > 6 weeks
- Most urticaria is acute and resolves

COMMON CAUSES OF ACUTE URTICARIA

- Idiopathic
- Infection
  - Upper respiratory, streptococcal infections, helminthes
- Food reactions
  - Shellfish, nuts, fruit, etc.
- Drug reactions
- IV administration
  - Blood products, contrast agents



ALLERGIC TRIGGERS

- **Acute Urticaria**
  - Drugs - β-lactam antibiotics, sulfonamides, aspirin
  - Foods - milk, eggs, peanuts, sesame, soy wheat, shellfish, fish
  - Food additives
  - Infections
  - Insect bites and stings
  - Contactants and inhalants (includes animal dander and latex)

Chronic Urticaria

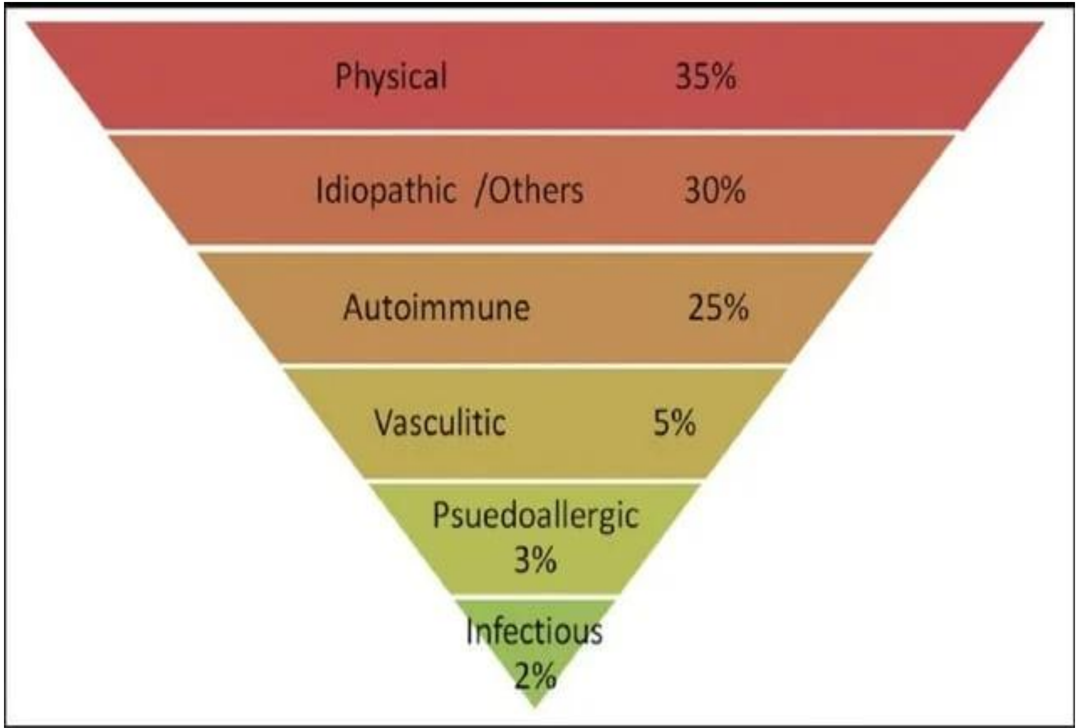
Physical factors

–cold
–heat
–dermatographic
–pressure
–solar

Idiopathic

ETIOLOGY OF CHRONIC URTICARIA

- Idiopathic: over 50% of chronic urticaria
- Physical urticarias: many patients with chronic urticaria have physical factors that contribute to their urticaria
  - These factors include pressure, cold, heat, water (aquagenic), sunlight (solar), vibration, and exercise
  - Cholinergic urticaria is triggered by heat and emotion
  - The diagnosis of pure physical urticaria is made when the sole cause of a patient's urticaria is a physical factor
- Chronic autoimmune: possibly a third or more of patients with chronic urticaria
- Other: infections, ingestions, medications



Physical 35%

Idiopathic /Others 30%

Autoimmune 25%

Vasculitic 5%

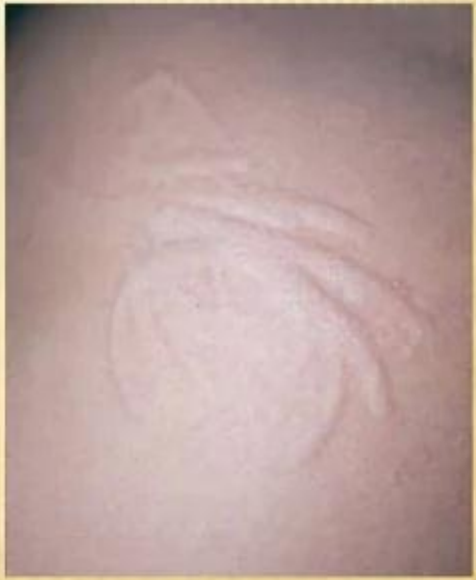
Psuedoallergic 3%

Infectious 2%

CHRONIC URTICARIA – COMMON CAUSES

DERMATOGRAPHISM

- Most common form of physical urticaria
- Sharply localized edema or wheal within seconds to minutes after the skin has been rubbed
- Affects 2-5% of the population

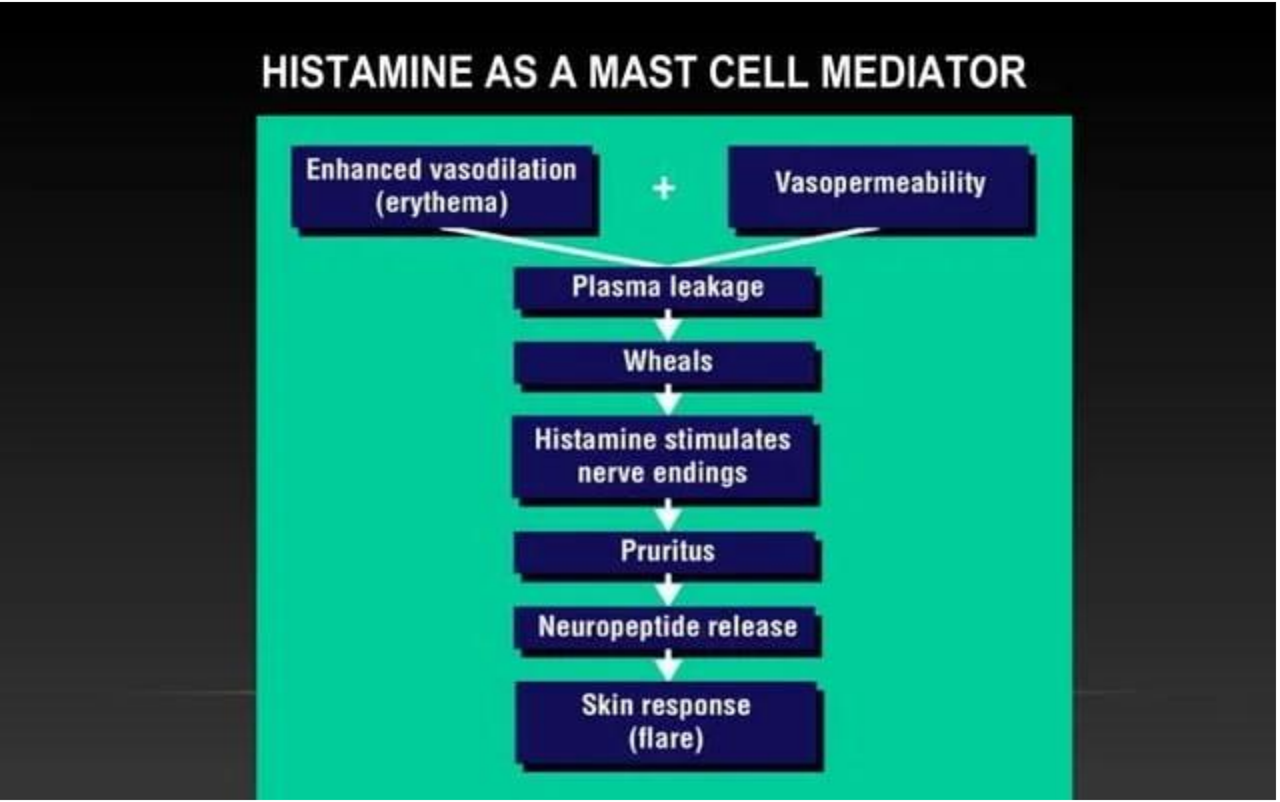


URTICARIA: PATHOPHYSIOLOGY

- The mast cell is the major effector cell in urticaria
- Immunologic Urticaria: antigen binds to IgE on the mast cell surface causing degranulation, which results in release of histamine
  - Histamine binds to H1 and H2 receptors to cause arteriolar dilatation, venous constriction and increased capillary permeability.

URTICARIA: PATHOPHYSIOLOGY

- Non-Immunologic Urticaria: not dependent on the binding of IgE receptors
  - For example, aspirin may induce histamine release through a pharmacologic mechanism where its effect on arachidonic acid metabolism causes a release of histamine from mast cells.
  - Physical stimuli may induce histamine release through direct mast cell degranulation



HISTAMINE AS A MAST CELL MEDIATOR

Enhanced vasodilation (erythema) + Vasopermeability

Plasma leakage

↓

Wheals

↓

Histamine stimulates nerve endings

↓

Pruritus

↓

Neuropeptide release

↓

Skin response (flare)

CLINICAL EVALUATION

- Ask about symptoms of anaphylaxis, including: chest tightness or difficulty breathing, hoarse voice or throat tightness, nausea, vomiting, abdominal pain, lightheadedness
- In addition to the skin exam, the physician should obtain a set of vital signs and evaluate for respiratory distress (dyspnea, wheeze, bronchospasm, stridor) and hypotension
- For acute urticaria, no lab testing is required
  - Laboratory testing is generally driven by associated signs and symptoms (e.g. C1 esterase deficiency only causes angioedema, not hives)

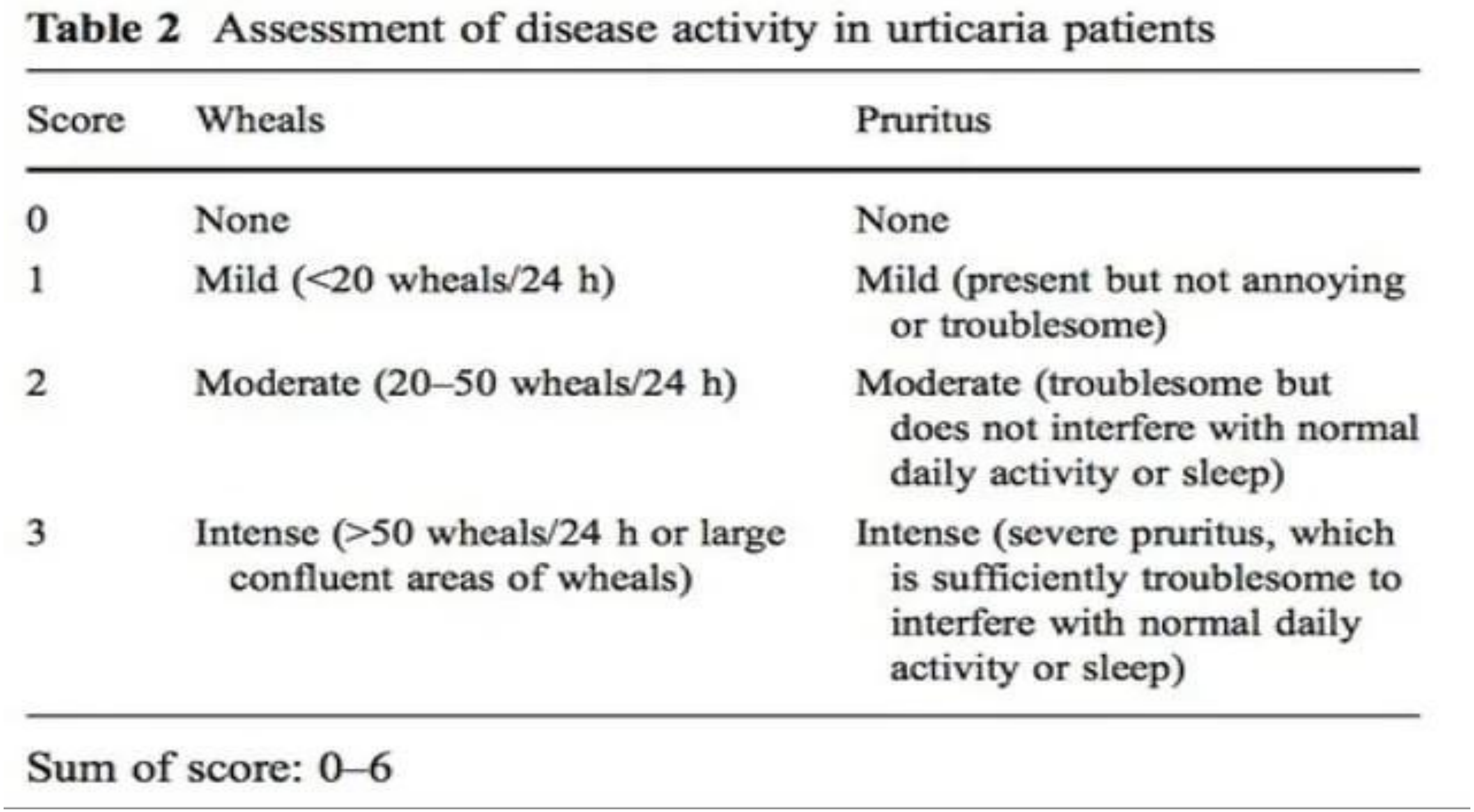


Table 2 Assessment of disease activity in urticaria patients

Score	Wheals	Pruritus
0	None	None
1	Mild (<20 wheals/24 h)	Mild (present but not annoying or troublesome)
2	Moderate (20–50 wheals/24 h)	Moderate (troublesome but does not interfere with normal daily activity or sleep)
3	Intense (>50 wheals/24 h or large confluent areas of wheals)	Intense (severe pruritus, which is sufficiently troublesome to interfere with normal daily activity or sleep)

Sum of score: 0–6

ALLERGY TESTING

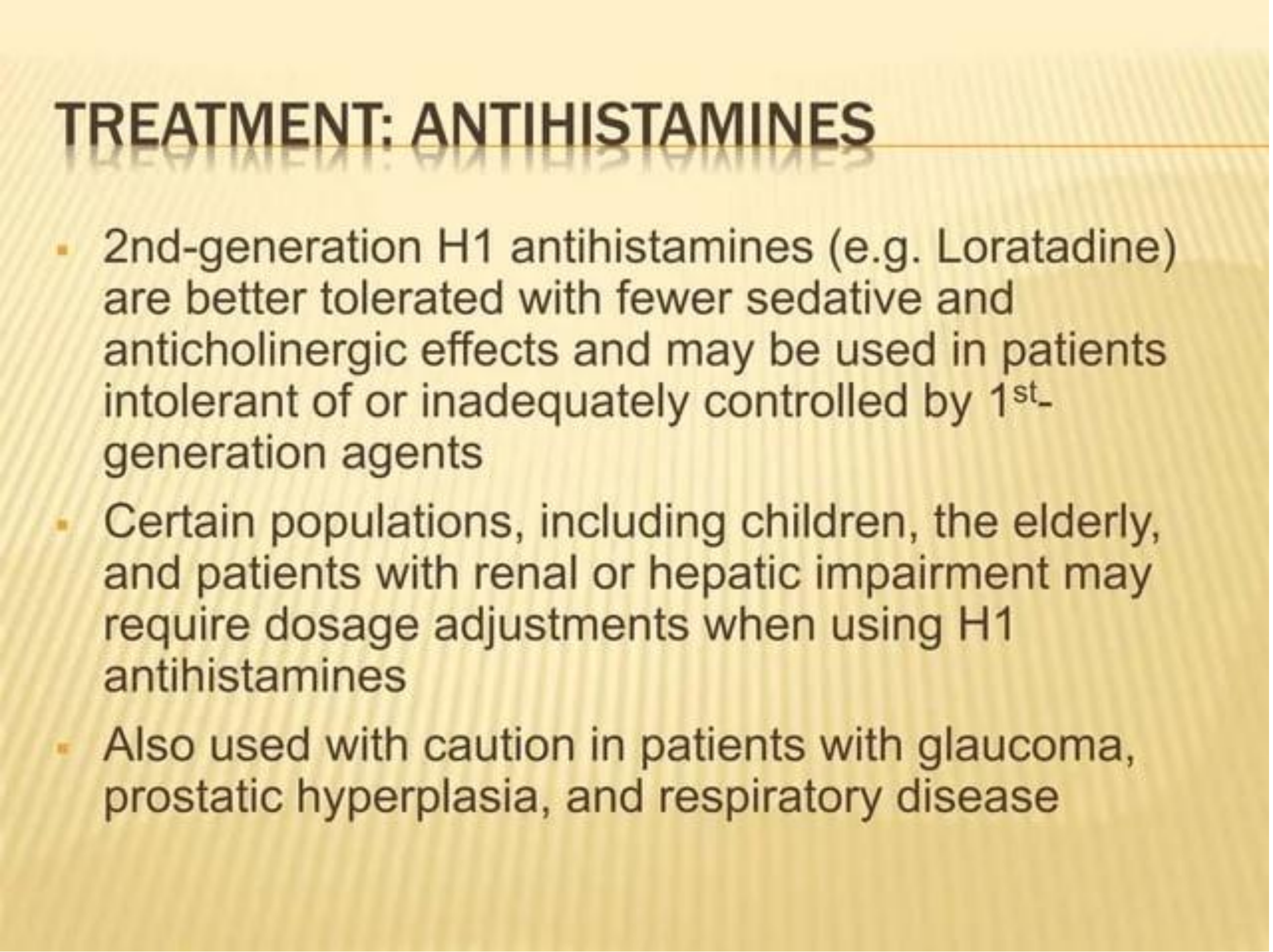
- Allergy testing is not routinely performed in patients with chronic urticaria.
  - Skin prick testing may reveal sensitivities to a variety of allergens that may not be relevant to the patient’s urticaria.
  - Laboratory tests may identify the 1/3 of patients with chronic urticaria who have an autoimmune pathogenesis. This adds additional costs and may not change the management.

NATURAL HISTORY AND PROGNOSIS

- Symptoms of chronic urticaria can be severe and impair the patient’s quality of life (QOL)
- In most patients, chronic urticaria is an episodic and self-limited disorder
- Average duration of disease is two to five years
- In patients with idiopathic chronic urticaria, there is a rate of spontaneous remission at one year of approximately 30 to 50 percent
- However, symptoms extend beyond five years in nearly one-fifth of patients

TREATMENT: ANTIHISTAMINES

- Oral H1 antihistamines are the first-line treatment for acute and chronic urticaria
- 1st-generation H1 antihistamines are less well-tolerated due to sedation
  - e.g. 10-50 mg hydroxyzine 1-2 hours before bedtime
  - Can start with smaller doses (10 mg) to allow the patient to manage the sedation effects
  - Remember to warn patient not to drive a car or operate other dangerous machines within 4-6 hours of taking this medication
  - Do not take with other sedating medications



TREATMENT: ANTIHISTAMINES

- 2nd-generation H1 antihistamines (e.g. Loratadine) are better tolerated with fewer sedative and anticholinergic effects and may be used in patients intolerant of or inadequately controlled by 1st-generation agents
- Certain populations, including children, the elderly, and patients with renal or hepatic impairment may require dosage adjustments when using H1 antihistamines
- Also used with caution in patients with glaucoma, prostatic hyperplasia, and respiratory disease

ANTIHISTAMINES

- The following are examples of H_1 antihistamines:
  - 1st Generation
    - Diphenhydramine (OTC)
    - Hydroxyzine (Rx, generic)
    - Chlorpheniramine (OTC)
  - 2nd Generation
    - Cetirizine (OTC)
    - Loratadine (OTC)
    - Fexofenadine (OTC)

BEYOND ORDINARY URTICARIA

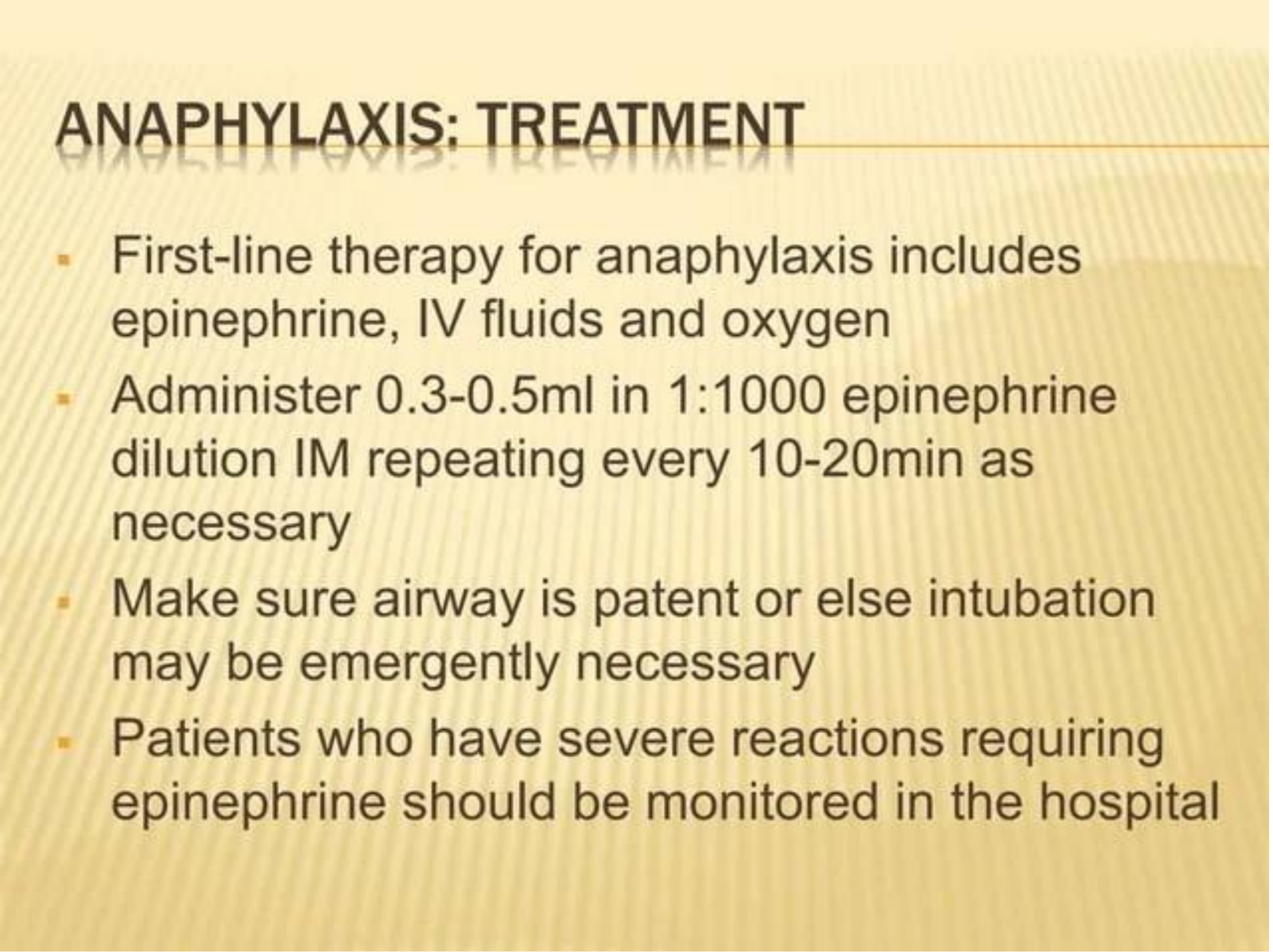
- The appearance of the hives does not tell you the underlying cause
- The presence of systemic symptoms should signal the possibility that an urticarial rash is not ordinary urticaria but rather a systemic syndrome with urticaria-like skin lesions

SYNOPSIS

- Urticaria (hives) is a vascular reaction of the skin characterized by wheals surrounded by a red halo or flare.
- Urticaria is classified as acute or chronic. Acute urticaria is defined as periodic outbreaks of urticarial lesions that resolve within six weeks.
- Over 50% of chronic urticaria is idiopathic.
- Oral H1 antihistamines are first-line treatment for acute and chronic urticaria.
- 1st generation H1 antihistamines can cause sedation.
- The presence of systemic symptoms should signal the possibility that an urticarial rash is not ordinary urtica

ANAPHYLAXIS

- Anaphylaxis is a serious allergic reaction that is rapid in onset and may cause death
- Patients with anaphylaxis may have no skin lesions, lesions of angioedema, and/or typical urticarial wheals
- Morphology of the skin lesion does not matter
    - Patients with angioedema are not more likely to have anaphylaxis compared to patients with urticaria
- ABC's first!
- Recruit more help. May need to triage to higher level of care (in clinic this means calling 911).



ANAPHYLAXIS: TREATMENT

- First-line therapy for anaphylaxis includes epinephrine, IV fluids and oxygen
- Administer 0.3-0.5ml in 1:1000 epinephrine dilution IM repeating every 10-20min as necessary
- Make sure airway is patent or else intubation may be emergently necessary
- Patients who have severe reactions requiring epinephrine should be monitored in the hospital

ECZEMA & ALLERGIC CONTACT DERMATITIS

Eczema-Dermatological Definition

- An acute, subacute but usually chronic pruritic inflammation of the epidermis and the dermis, often occurring in association with a personal family history of hay fever, asthma, allergic rhinitis or atopic dermatitis. $_1$
- 1 Color Atlas and Synopsis of Clinical Dermatology

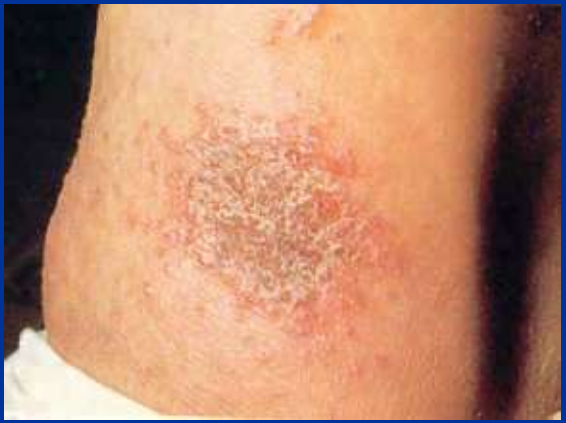
Characteristics of Acute **Eczema**

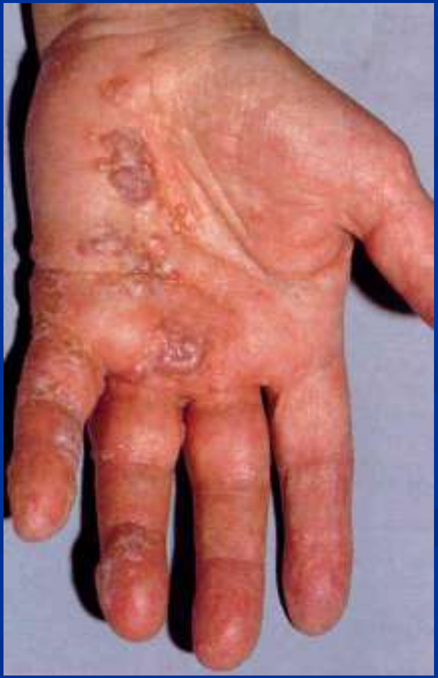
- Well demarcated plaques of **erythema and edema** on which are superimposed and closely spaced small vesicles filled with clear fluid with punctate erosions and crusting
- Distribution may be isolated and localized or general

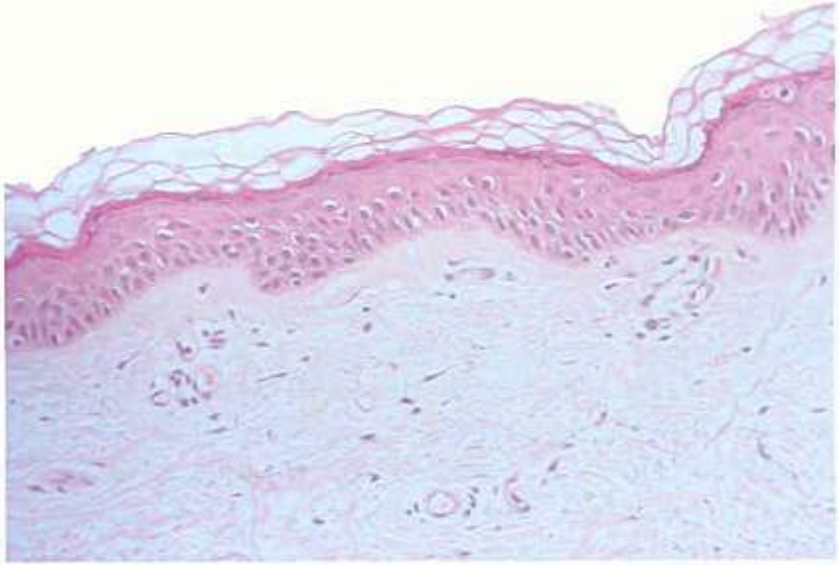
Acute **Eczema**

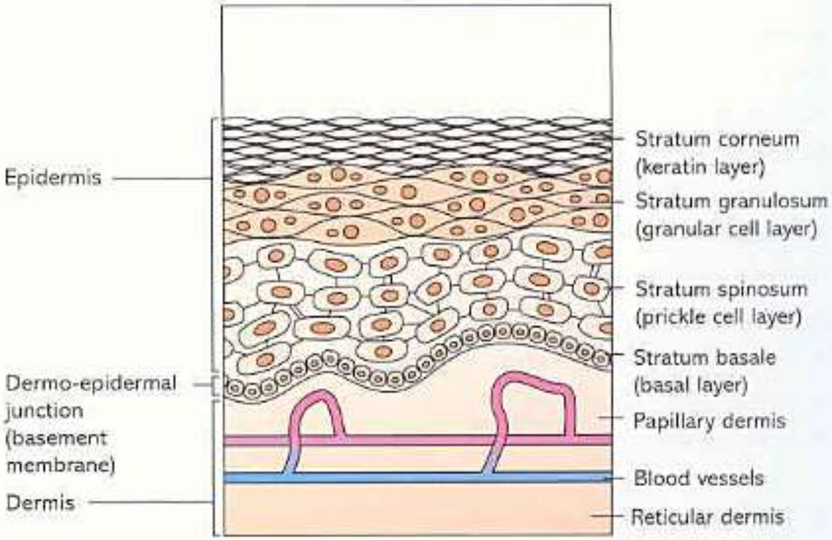
(Note the erythema, vesicles and swelling)

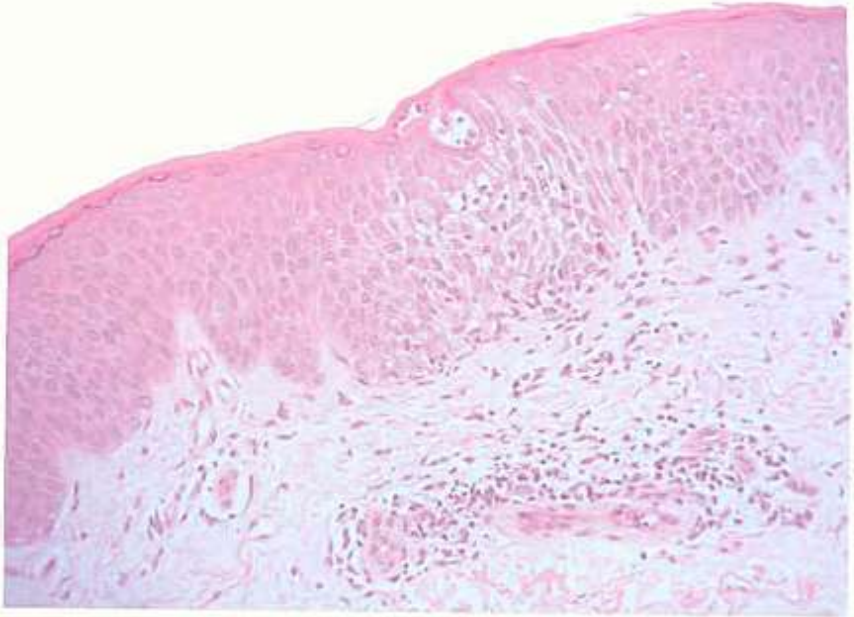
- Term dyshidrotic is a misnomer as sweat glands are not involved
- Also known as pompholyx

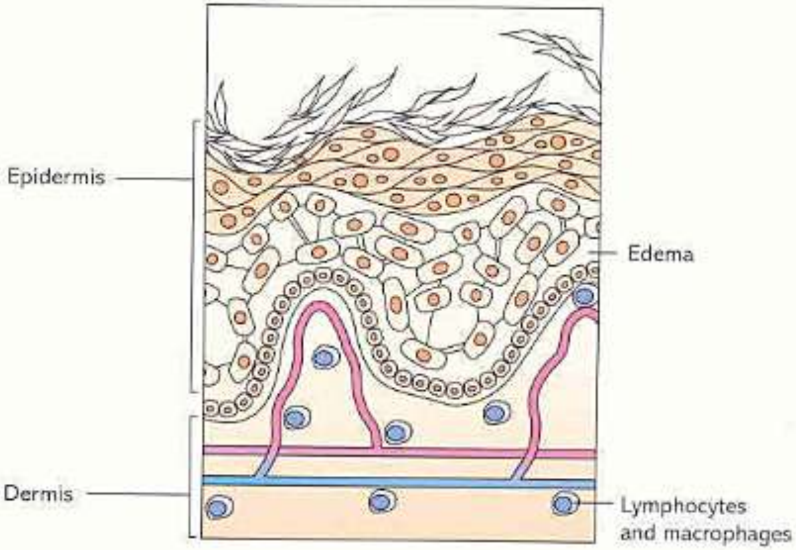







Epidermis
Dermo-epidermal junction (basement membrane)
Dermis
Stratum corneum (keratin layer)
Stratum granulosum (granular cell layer)
Stratum spinosum (prickle cell layer)
Stratum basale (basal layer)
Papillary dermis
Blood vessels
Reticular dermis



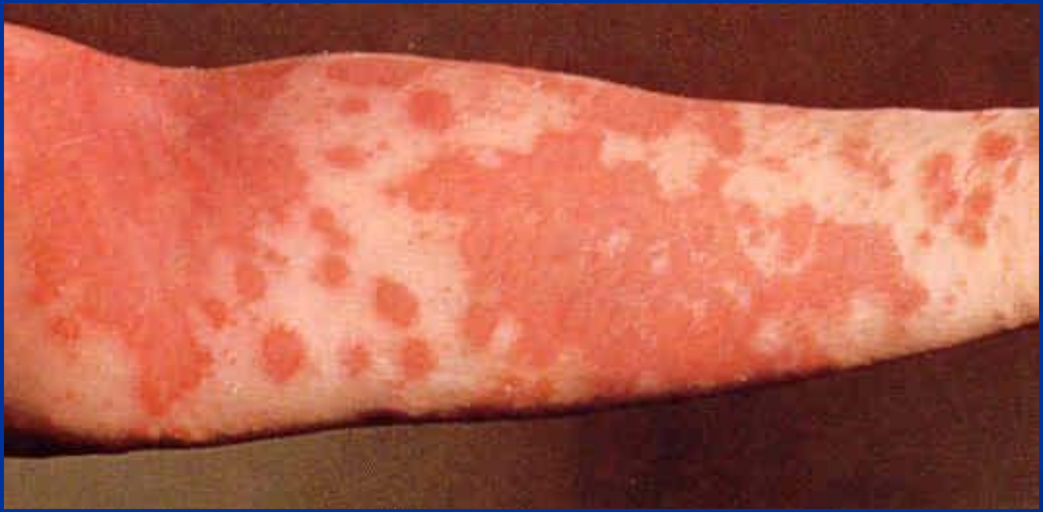

Epidermis
Dermis
Edema
Lymphocytes and macrophages

Characteristics of Subacute Eczema

- Plaques of mild erythema with small dry scales and or superficial desquamation, sometimes associated with small red, pointed or round papules
- Distribution may be isolated and localized or general

Subacute **Eczema**

- Note erythema, swelling and desquamation



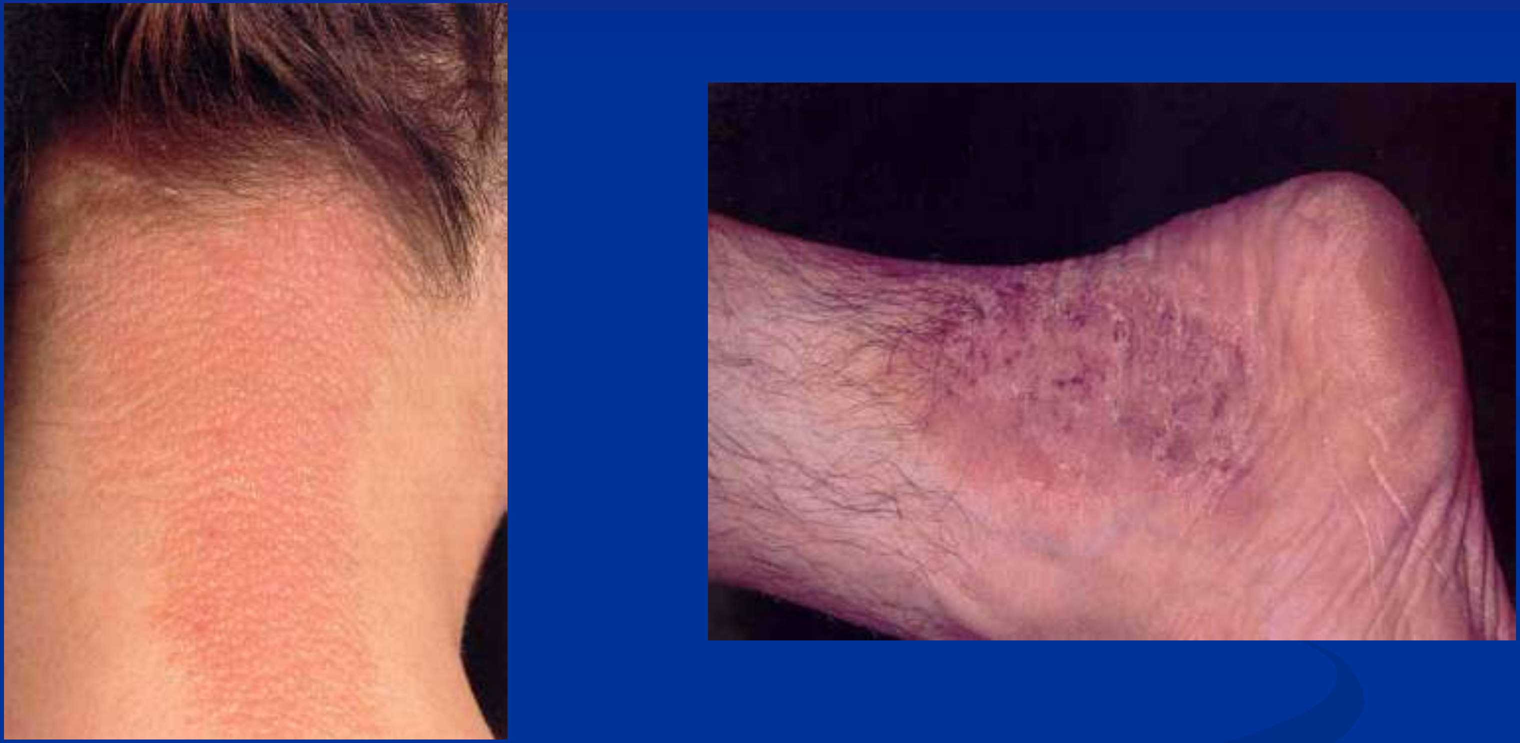
Characteristics of Chronic **Eczema**

- Plaques of **lichenification** with deepening of the skin lines with satellite, small, firm flat or round top papules, excoriations and pigmentations or mild erythema

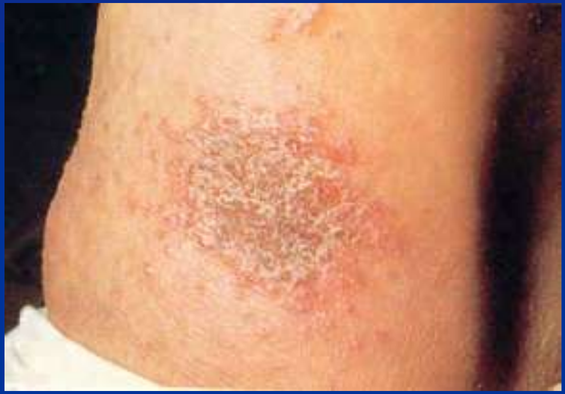
  Distribution – isolated and localized or generalized

Chronic **Eczema**

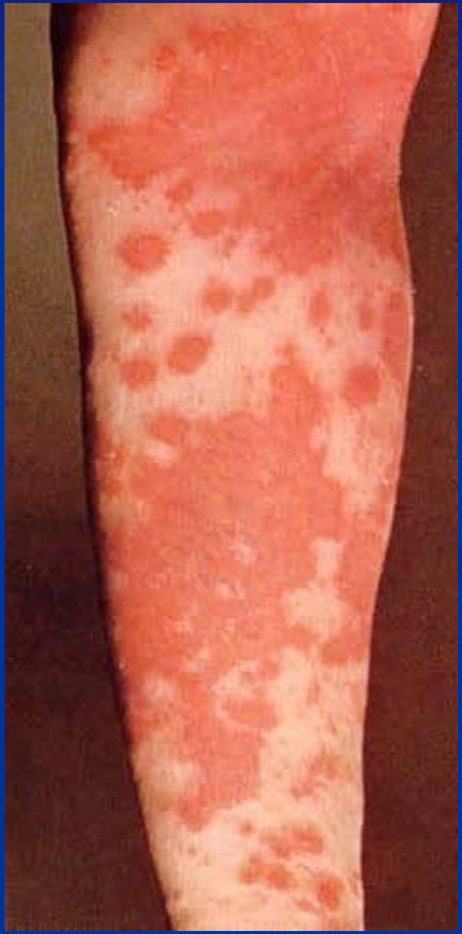
- Note lichenification, scaling and fissuring



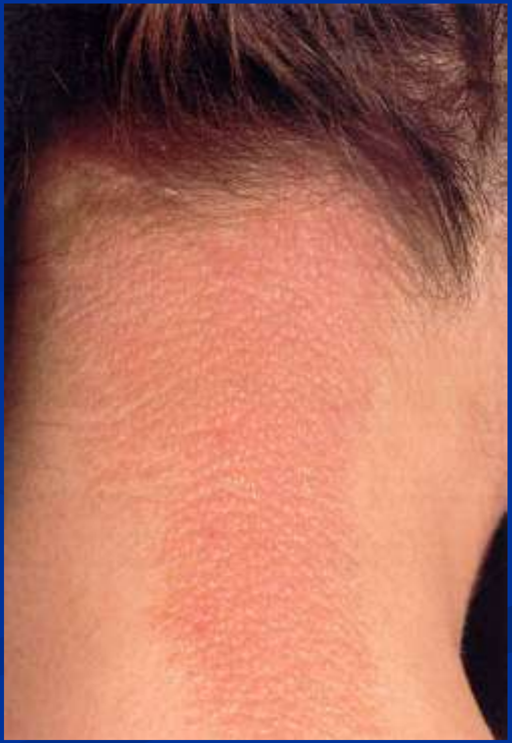
Acute - Subacute - Chronic



Swelling and erythema



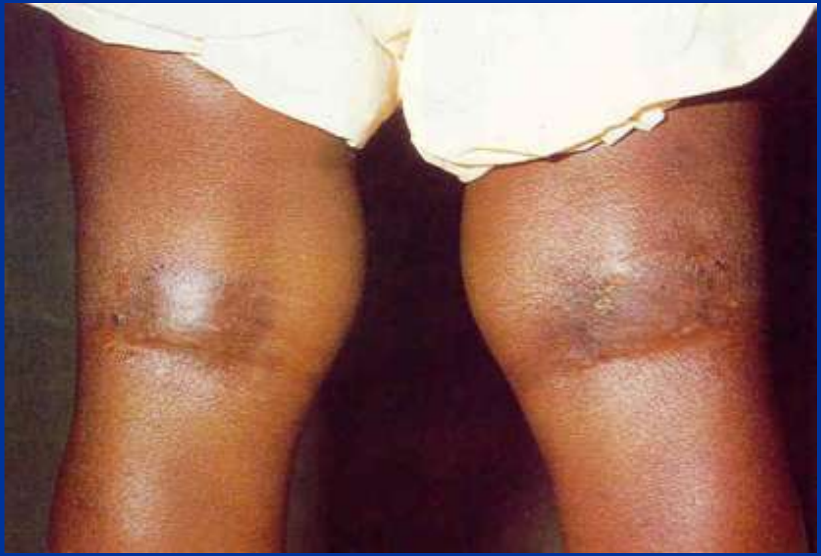
Punctate erythema, desquamation

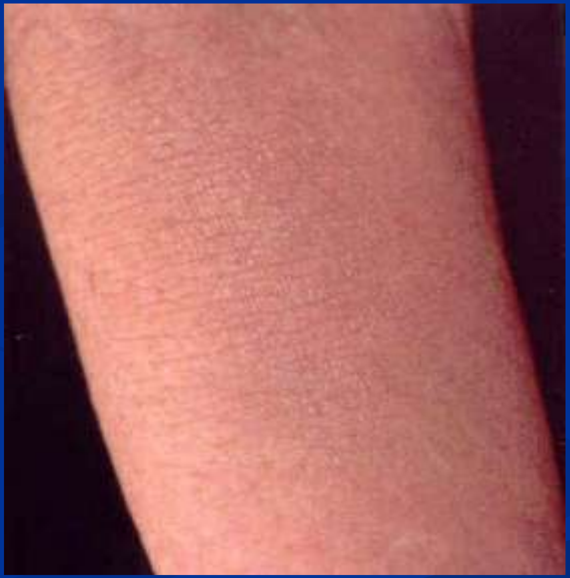


Lichenification

Acute, Subacute or Chronic?

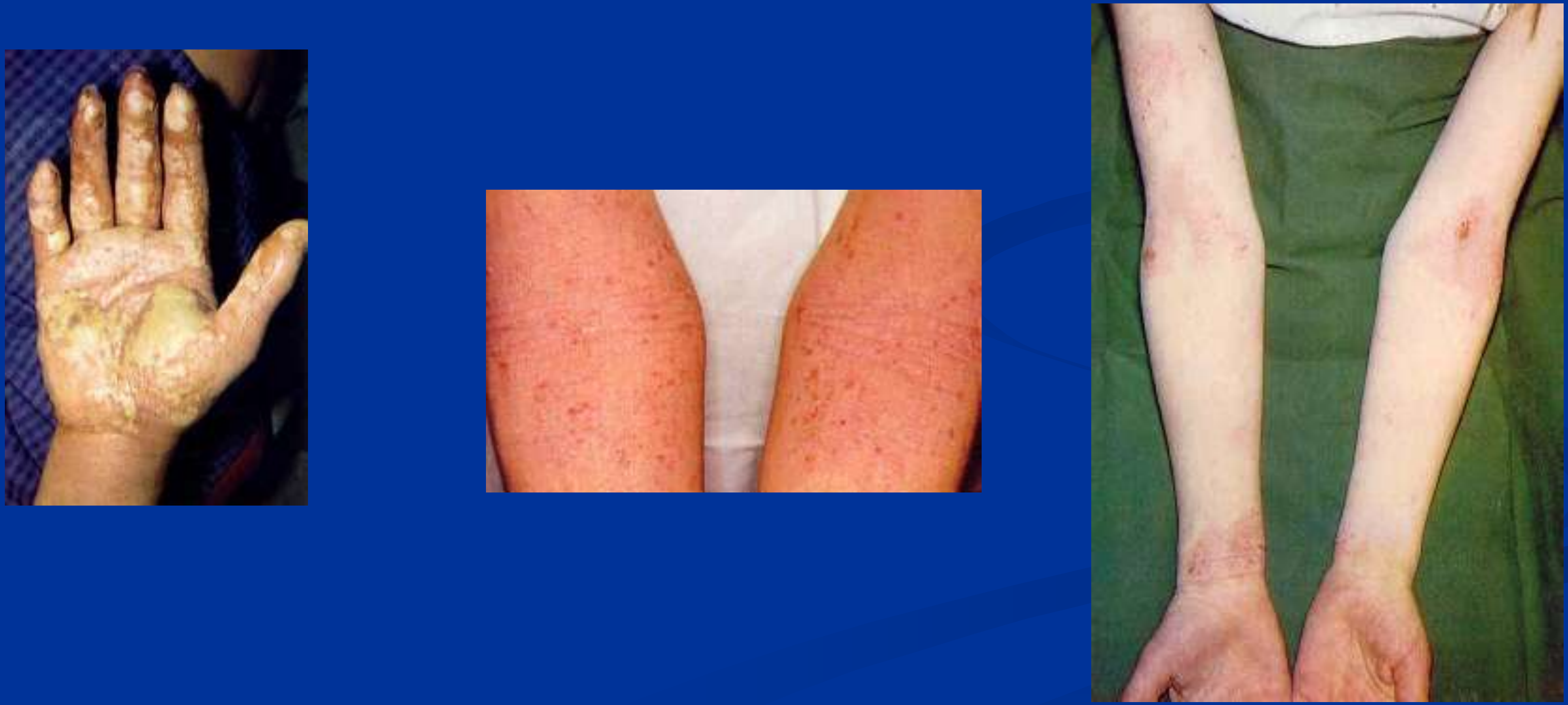
- Check for erythema, swelling, desquamation, lichenification





Acute, Subacute or Chronic?

- Check for erythema, swelling, desquamation, lichenification



Classification of Eczema/Dermatitis

Historically

- **Endogenous (occurring from within) dermatitis** was given the name “**eczema**”

- **Exogenous dermatitis (occurring from without)** was termed “**dermatitis**”

Classifications of Eczema

Endogenous

- Atopic or IgE
- Seborrheic
- Discoid or nummular
- Pompholyx
- Venous
- Asteatotic
- Juvenile plantar
- Erythoderma

Exogenous

- Allergic
- Toxic irritant contact
- Photosensitive

Atopic/IgE Eczema (endogenous or exogenous?)

Characteristics:

- 60% have onset in the first year of life
- Influenced by genetics and environmental factors
- More common in males that females
- Ethnicity may be a factor –less common in Asians; more common in Westerners and higher socioeconomic families
- Theory is - manifestation of well nourished immune system rarely challenged by infection
- Rare to have adult onset
- 2/3 of patients have family history of asthma, hay fever or allergic rhinitis

Atopic/IgE Eczema cont.

- **Characteristics:**
- May persist months to years
- All patients have dry skin
- Exacerbations caused by allergens, stress, hormones, climate, skin dehydration
- Physical characteristic may include all phases

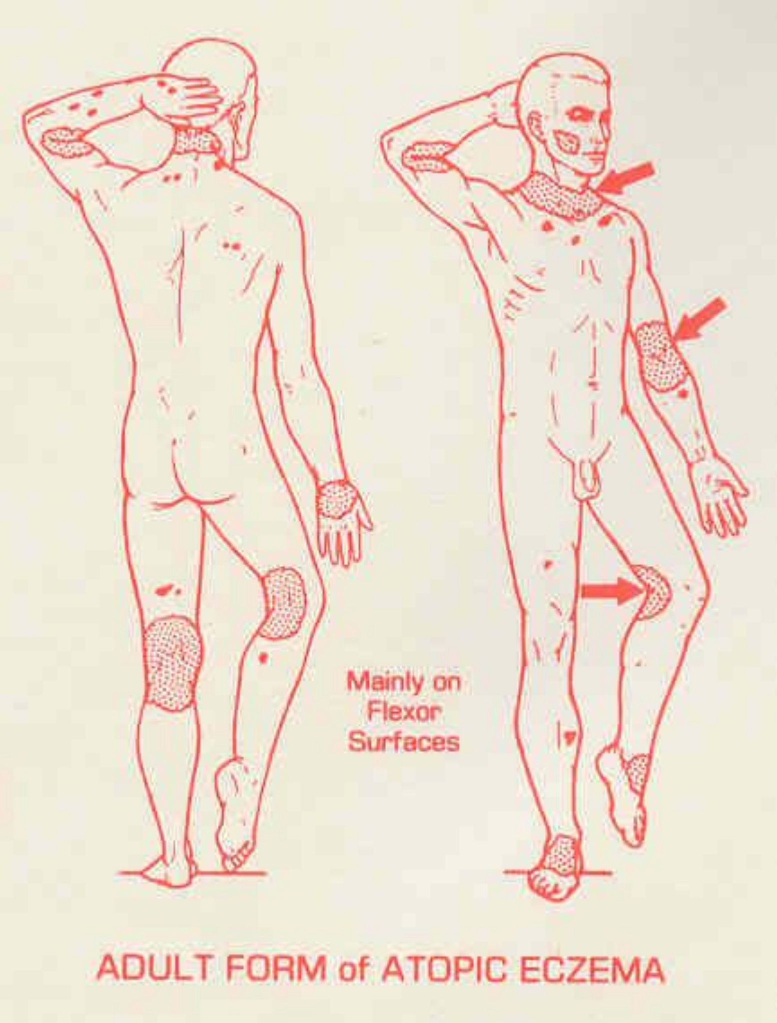
Distinctive Characteristics:

- Lesions are usually bilateral
- Located frequently in skin folds/creases and flexor surfaces

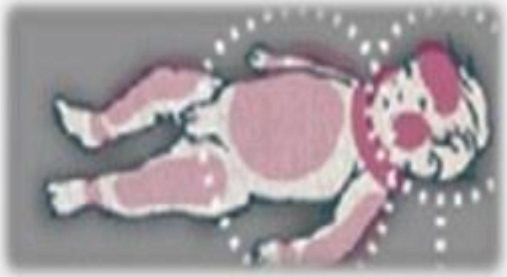
Atopic/IgE Eczema Distribution

Note:

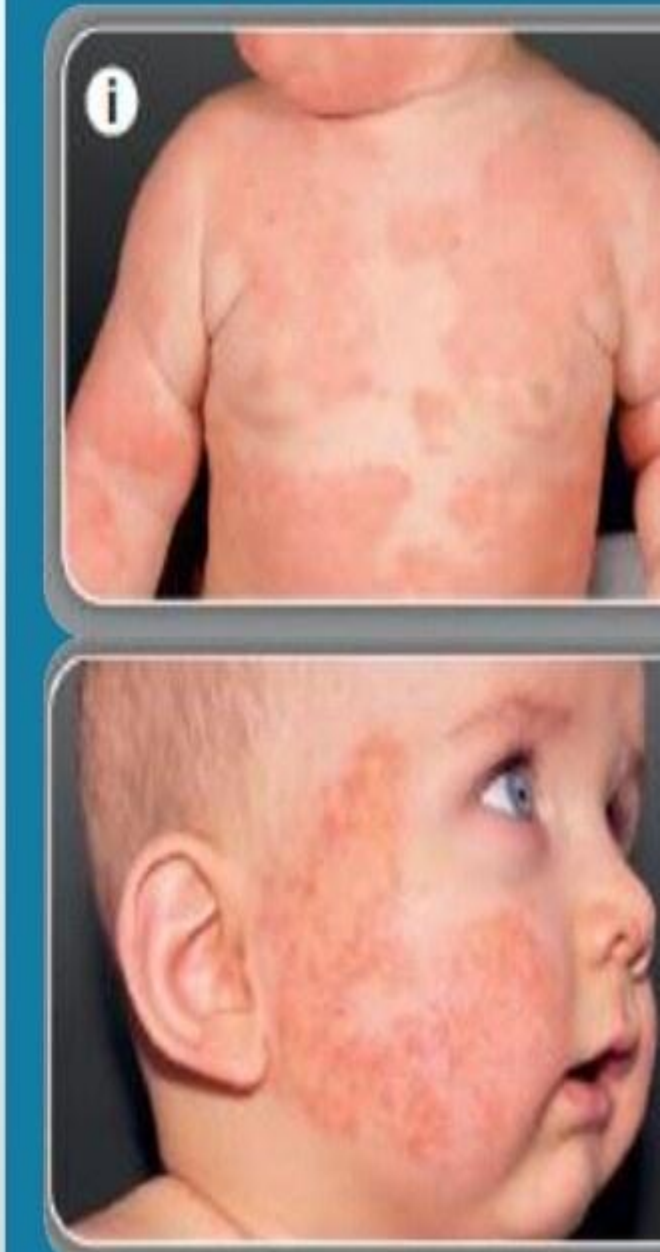
- Bilateral
- Skin folds and flexor surfaces


Mainly on Flexor Surfaces
ADULT FORM of ATOPIC ECZEMA

Infants

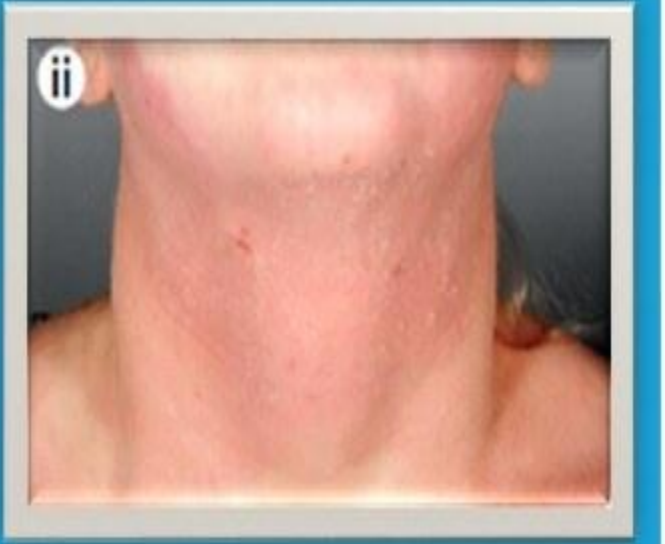


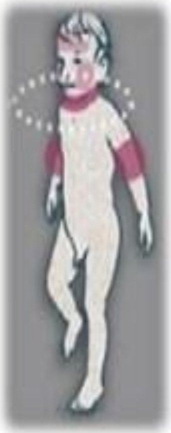
- Infants often present with acute lesions.
- Poorly defined erythema with edema, vesicles, excoriations, and serous exudate.
- Typically involvethe face and the extensor surfaces of the limbs.
- The trunk might be affected but the nappy area is typically spared.



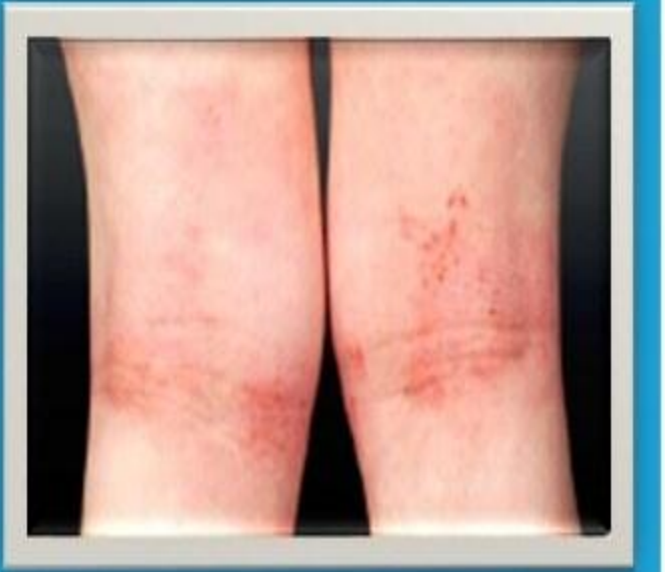
Childhood

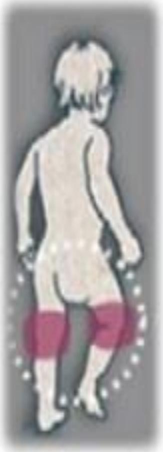
(aged 2 years and older)





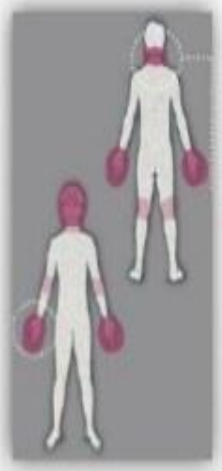
- Eczema becomes more localised and chronic than in infancy.



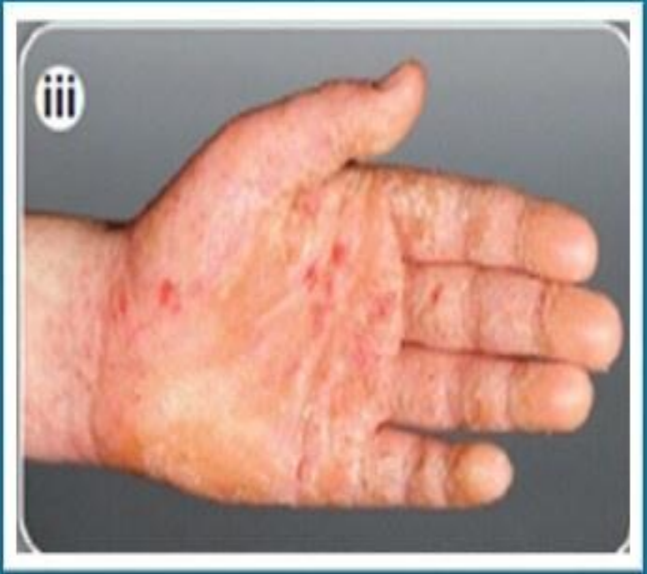


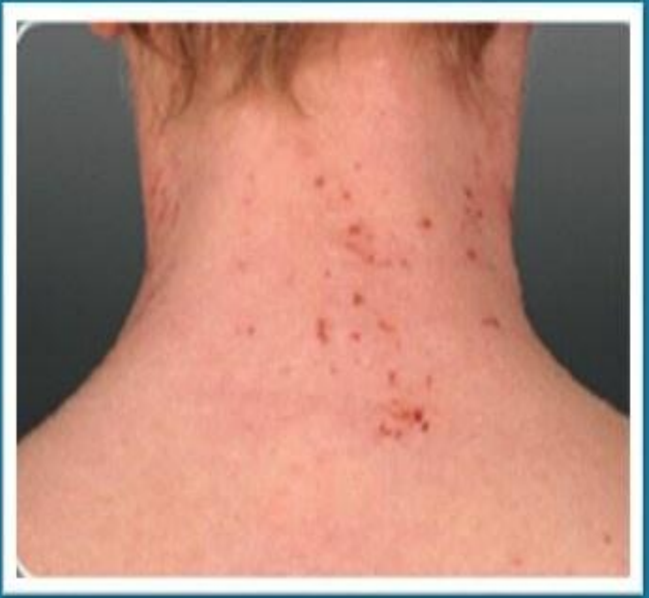
- Paler erythema, dry skin (xerosis), poorly defined lesions
- Commonly affecting flexor surfaces with thickening (lichenification) of chronic areas.

Adults



- Adolescents and adults usually have diffuse eczema.
- They can also have localised lesions typically affecting hands, eyelids, and flexures.
- Adults can have chronic hand eczema only or head-neck dermatitis as well, involving the upper trunk, shoulders, and scalp.

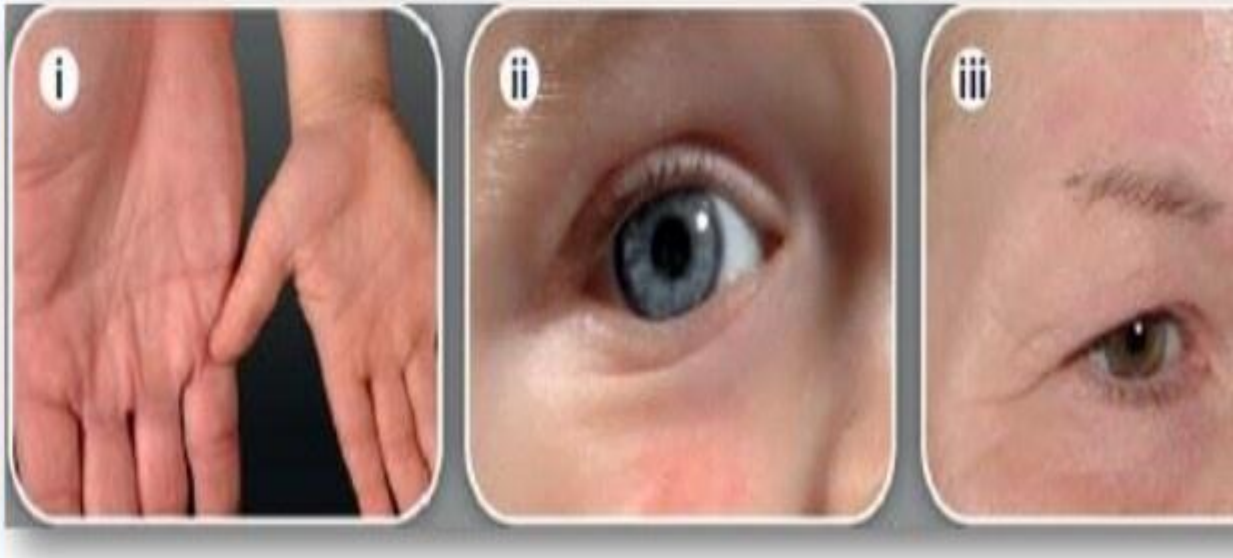




Langan SM, Irvine AD, Weidinger S. Atopic dermatitis. Lancet. 2020;396(10247):345–60.

Associated non-specific features

- **Hyperlinearity of palms and soles**
  : mostly seen in patients with *FLG* mutations or ichthyosis vulgaris
- **Dennie-Morgan infraorbital folds**
- **Hertoghe's sign.**



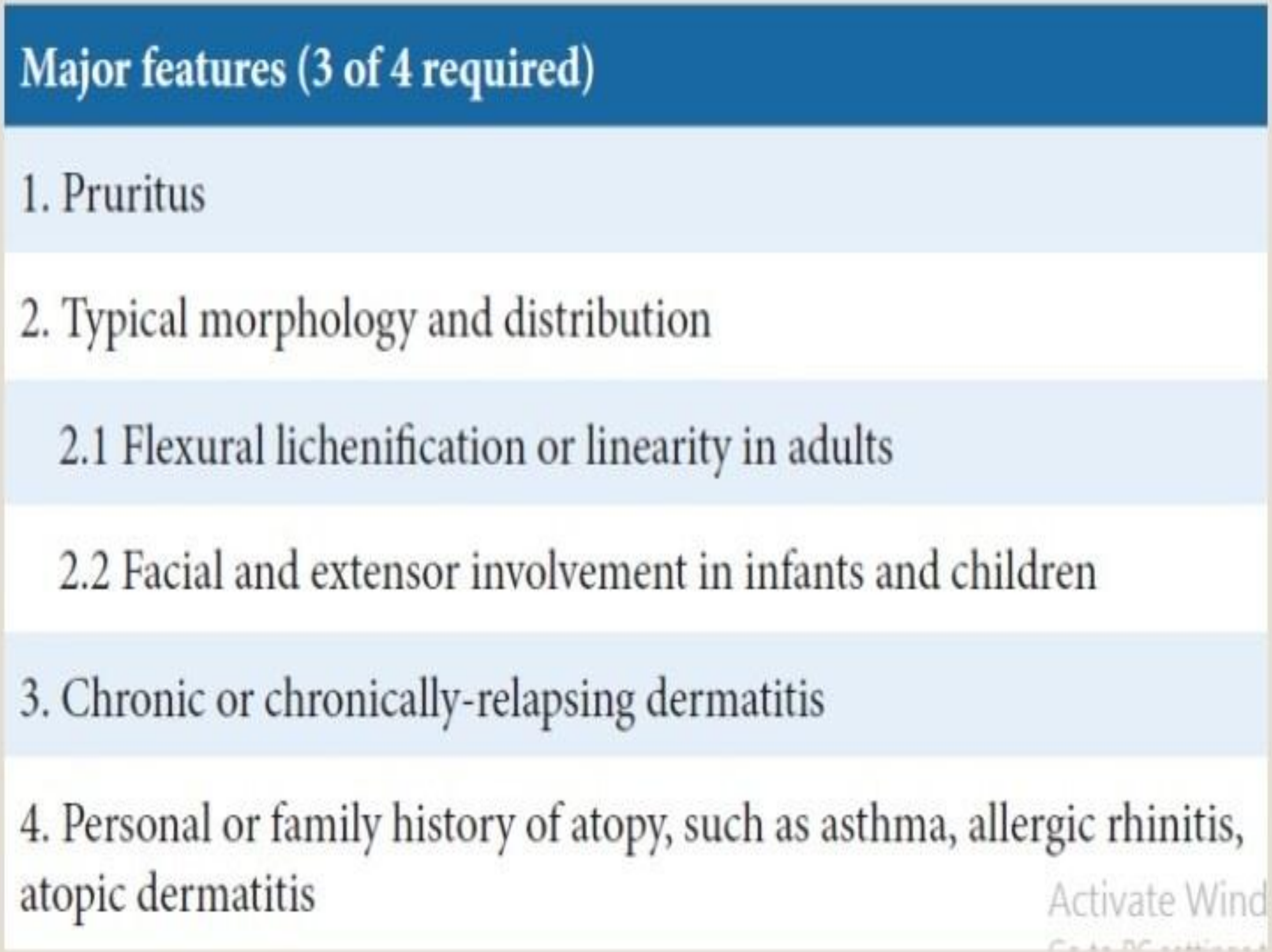
Diagnostic

- The diagnosis of AD is based on one’s history and clinical manifestations, such as morphology and the distribution of skin lesions.

- In 1980, major and minor criteria for a diagnosis of AD were proposed by Hanifin and Rajka.

- Three of four major criteria and three of 23 minor criteria are required for a diagnosis of AD.

Features to be considered in the diagnosis of patients with atopic dermatitis by Hanifin & Rajka (1980)



Major features (3 of 4 required)
1. Pruritus
2. Typical morphology and distribution
2.1 Flexural lichenification or linearity in adults
2.2 Facial and extensor involvement in infants and children
3. Chronic or chronically-relapsing dermatitis
4. Personal or family history of atopy, such as asthma, allergic rhinitis, atopic dermatitis

Activate Wind

Minor features (3 of 23 required)

1. Xerosis
2. Ichthyosis/palmar hyperlinearity/keratosis pilaris
3. Immediate (type 1) skin test reactivity
4. Elevated serum immunoglobulin E
5. Early age of onset
6. Tendency toward cutaneous infections (S. *aureus* and Herpes simplex virus)/impaired, cell-mediated immunity
7. Tendency toward non-specific hand or foot dermatitis
8. Nipple eczema
9. Chelitis
10. Recurrent conjunctivitis
11. Dennie-Morgan infraorbital fold
12. Keratoconus
13. Anterior subcapsular cataract
14. Orbital darkening
15. Facial pallor/facial erythema
16. Pityriasis alba
17. Anterior neck fold
18. Itch when sweating
19. Intolerance to wool and lipid solvents
20. Perifollicular accentuation
21. Food intolerance
22. Course influence by environmental/emotional factors
23. White dermographism/delayed blanch

Atopic/IgE Eczema cont.

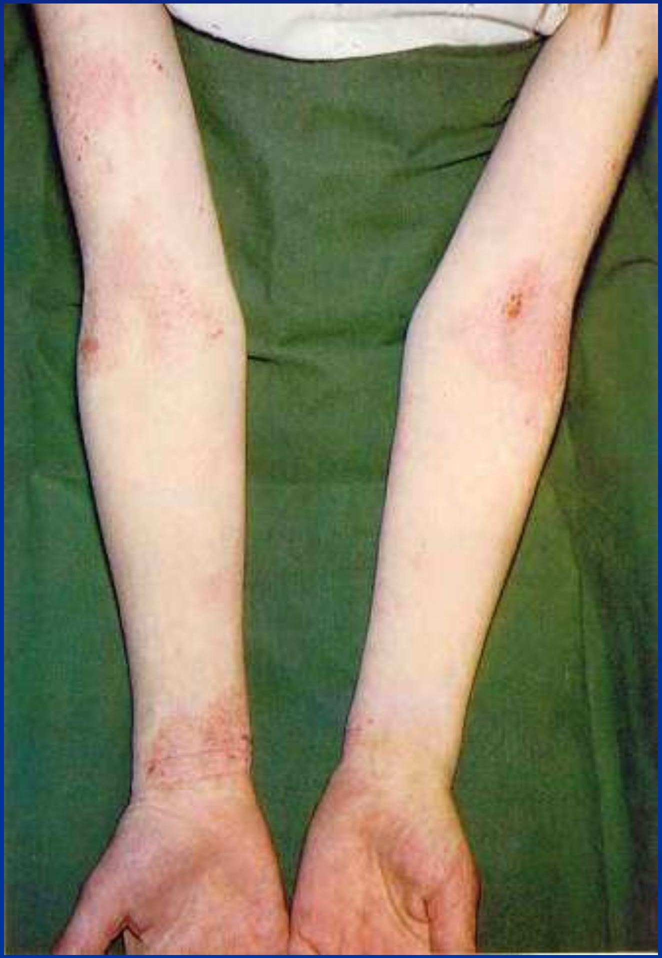
Confused with:

- Scabies, seborrhea, psoriasis and, contact dermatitis

Atopic/IgE Eczema cont.

- **Treatment:**
- Avoid scratching, clean and cool environment, use of soap substitutes
- Emollients
- Topical steroids
- Topical immunomodulators –tacrolimus
- Systemic antihistamines
- Soaks
- Tar preparations

Atopic/IgE Dermatitis



Allergic (Contact)Eczema (exogenous or endogenous?)

Characteristic:

- Delayed, cell mediated hypersensitivity
- Strong sensitizer results in reaction soon after exposure
- Weak sensitizer my take months or years to develop reaction
- Age does not influence capacity for sensitization but more common in adults
- Black skin is less susceptible
- Important cause of disability in industry
- Non seasonal

Allergic (Contact) Eczema cont.

Characteristics:

- usually clears quite rapidly on withdrawal of offending agent
- may appear as erythematous papules, vesicles or bullous
- more common where epidermis is thinner

Distinctive Characteristics:

- Initial lesions usually limited to contact area
- not bilateral
- lesions with sharp borders or angles are pathognomonic

Causes of Allergic/Contact Eczema

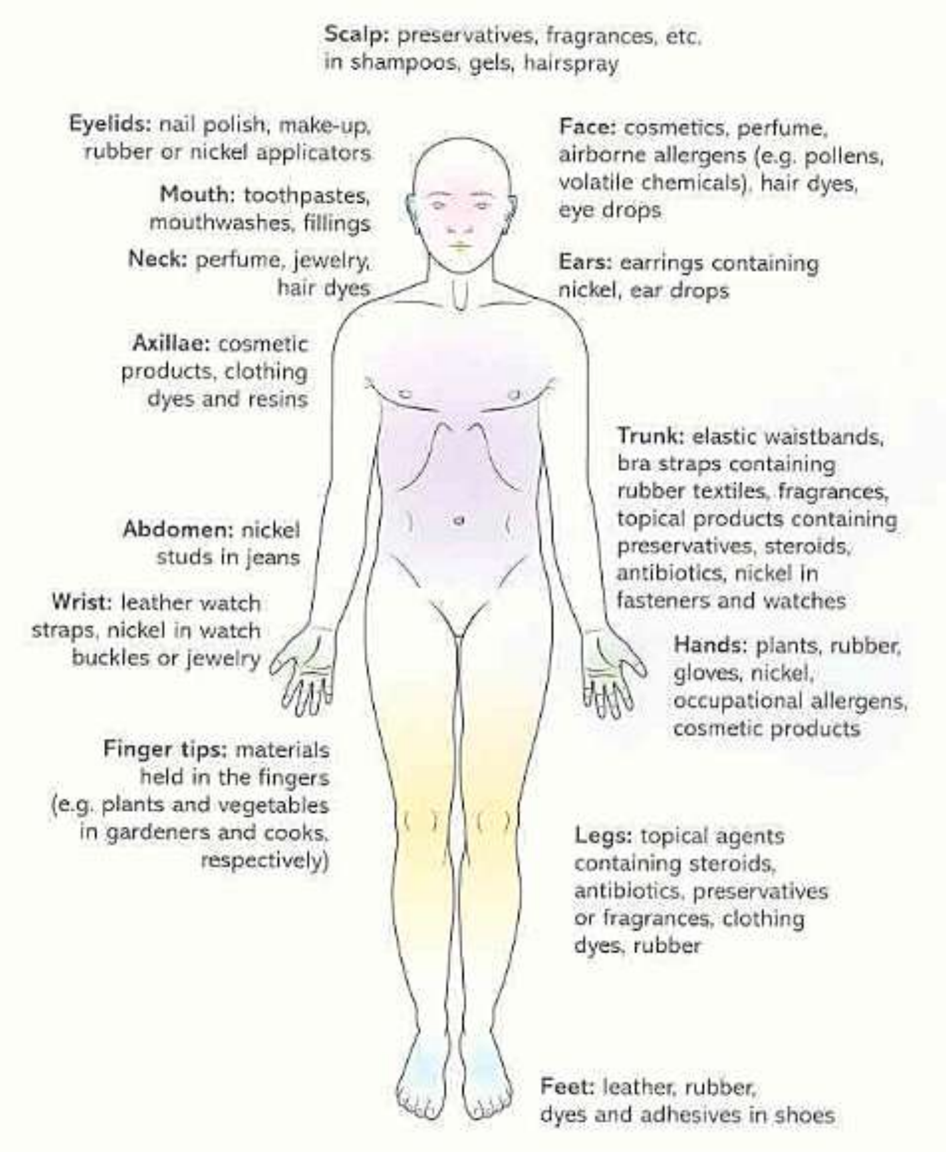
- Metals- nickel, platinum (10% of women)
- Detergents
- Plants and fibers
- Chemicals and dyes
- Polyethylene glycol and polysorbate 60
- Topical antibiotics and medications
- Animal keratin

Allergic/Contact Eczema

cont.

- **Treatment** – remove causative agent, Burow’s soaks 1:40, or saline 1tsp/pt warm water, Aveeno or oatmeal baths, calamine
- Systemic antihistamines
- Topical steroids, oral steroid taper
- Antibiotics for secondary infection
- **Confused with** – Atopic eczema, seborrhea, HSV

Allergic/ Contact Eczema Distribution



Scalp: preservatives, fragrances, etc. in shampoos, gels, hairspray

Eyelids: nail polish, make-up, rubber or nickel applicators

Face: cosmetics, perfume, airborne allergens (e.g. pollens, volatile chemicals), hair dyes, eye drops

Mouth: toothpastes, mouthwashes, fillings

Neck: perfume, jewelry, hair dyes

Ears: earrings containing nickel, ear drops

Axillae: cosmetic products, clothing dyes and resins

Trunk: elastic waistbands, bra straps containing rubber textiles, fragrances, topical products containing preservatives, steroids, antibiotics, nickel in fasteners and watches

Abdomen: nickel studs in jeans

Wrist: leather watch straps, nickel in watch buckles or jewelry

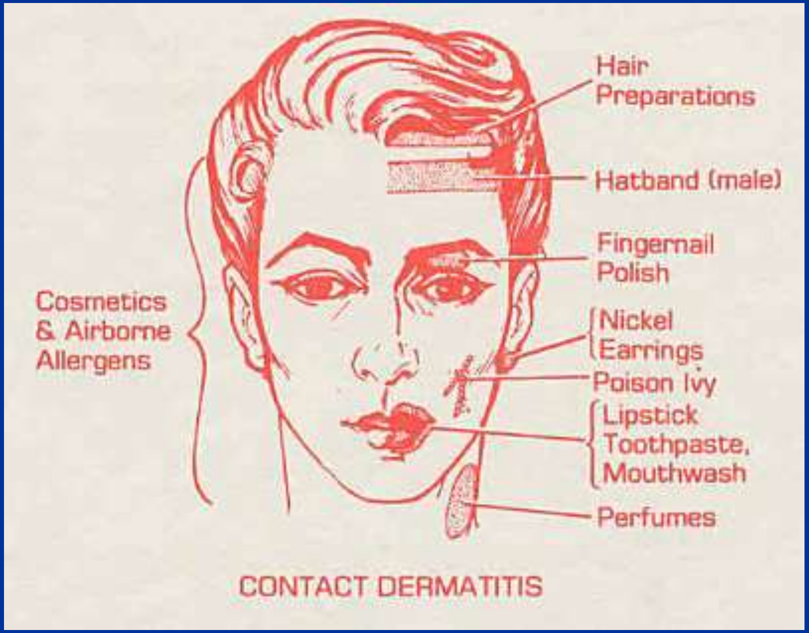
Hands: plants, rubber, gloves, nickel, occupational allergens, cosmetic products

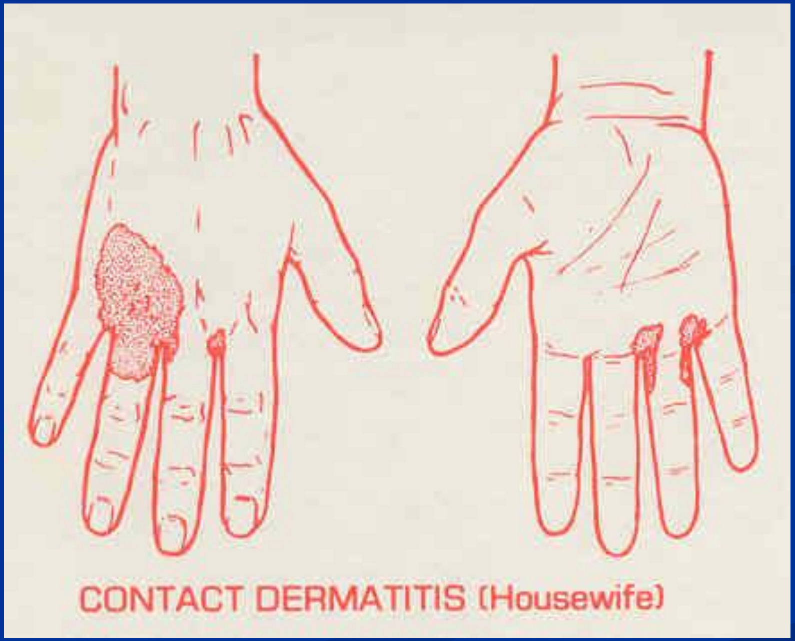
Finger tips: materials held in the fingers (e.g. plants and vegetables in gardeners and cooks, respectively)

Legs: topical agents containing steroids, antibiotics, preservatives or fragrances, clothing dyes, rubber

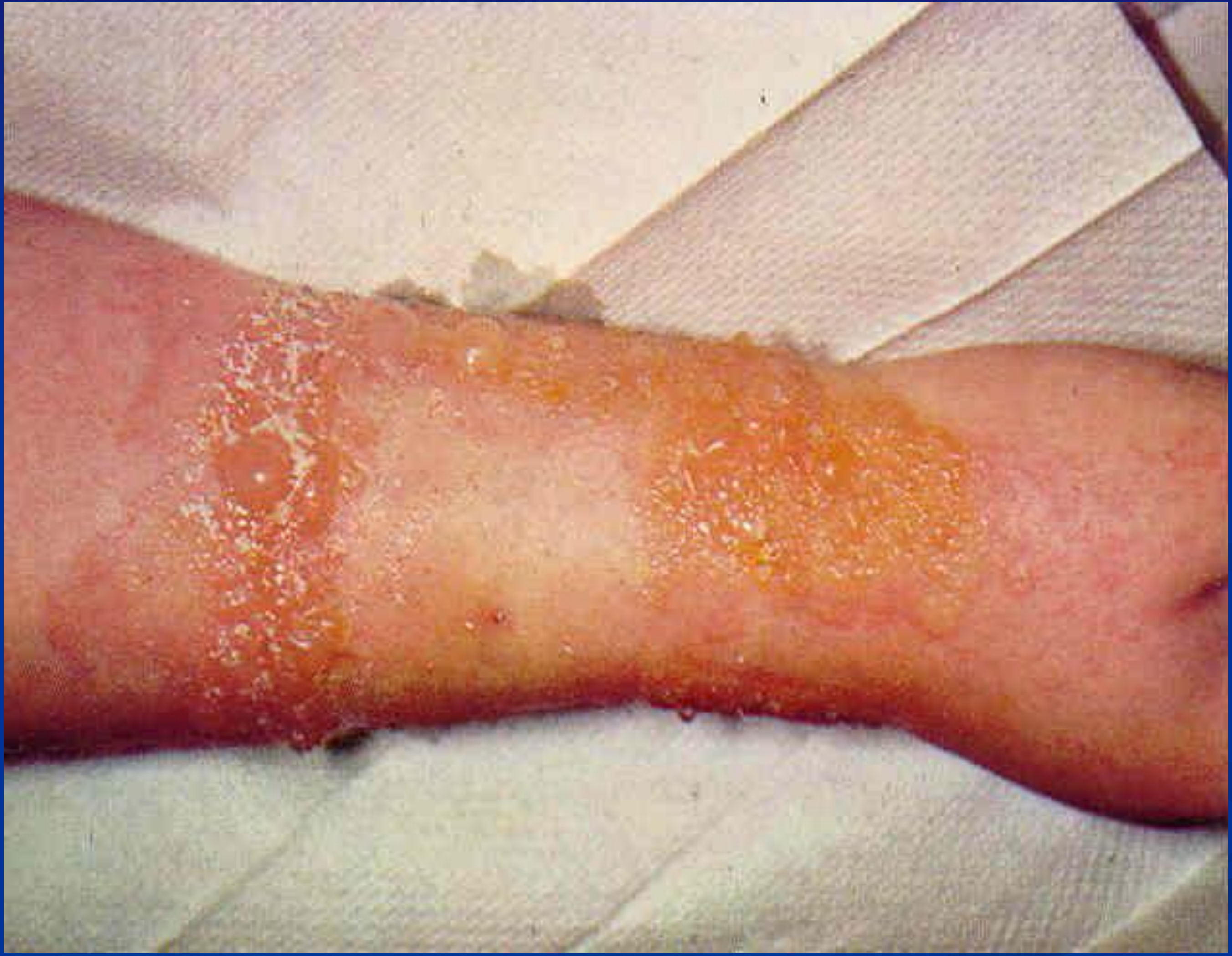
Feet: leather, rubber, dyes and adhesives in shoes

Allergic/ Contact Eczema Distribution


Hair Preparations
Hatband (male)
Fingernail Polish
Nickel Earrings
Poison Ivy
Lipstick Toothpaste, Mouthwash
Perfumes
Cosmetics & Airborne Allergens
CONTACT DERMATITIS

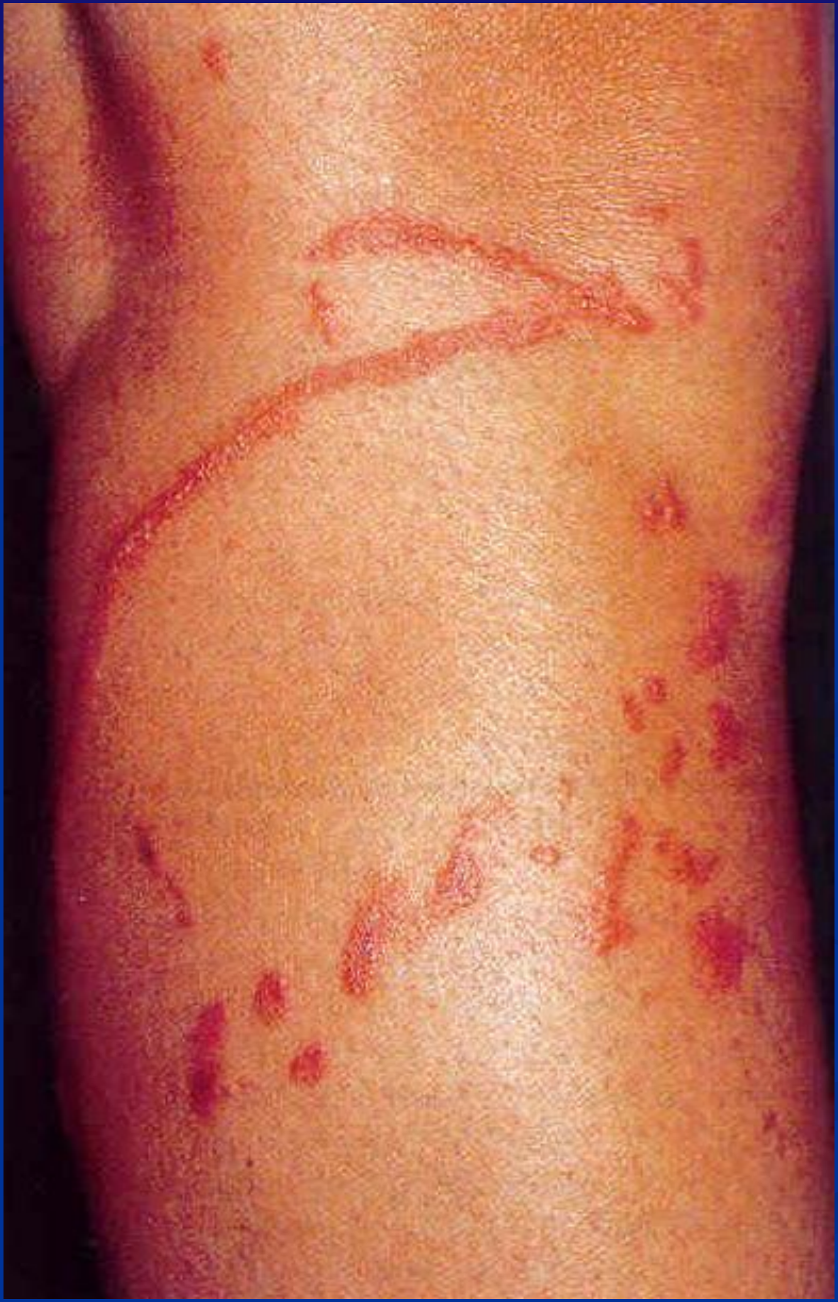

CONTACT DERMATITIS (Housewife)

Note: distribution

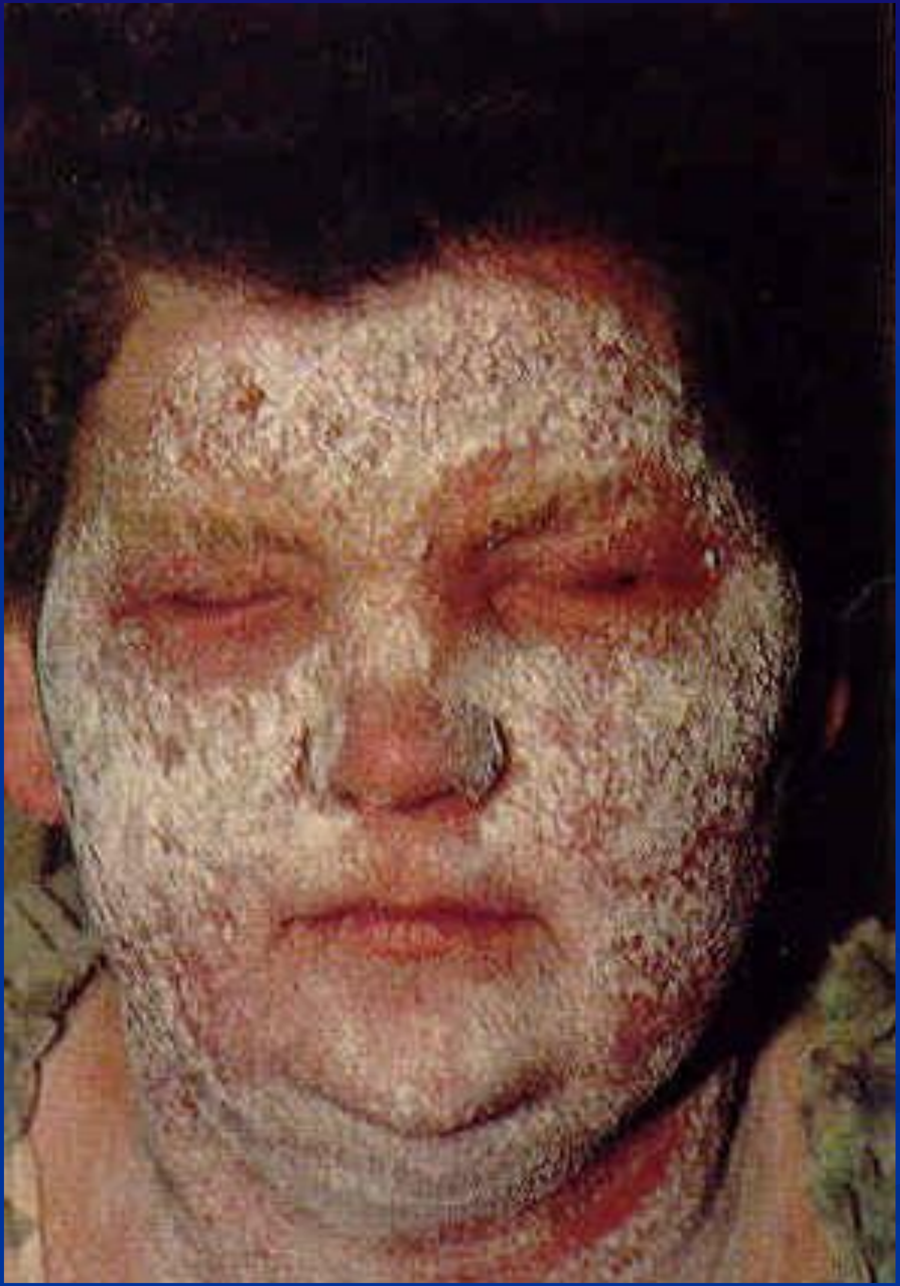


Note:

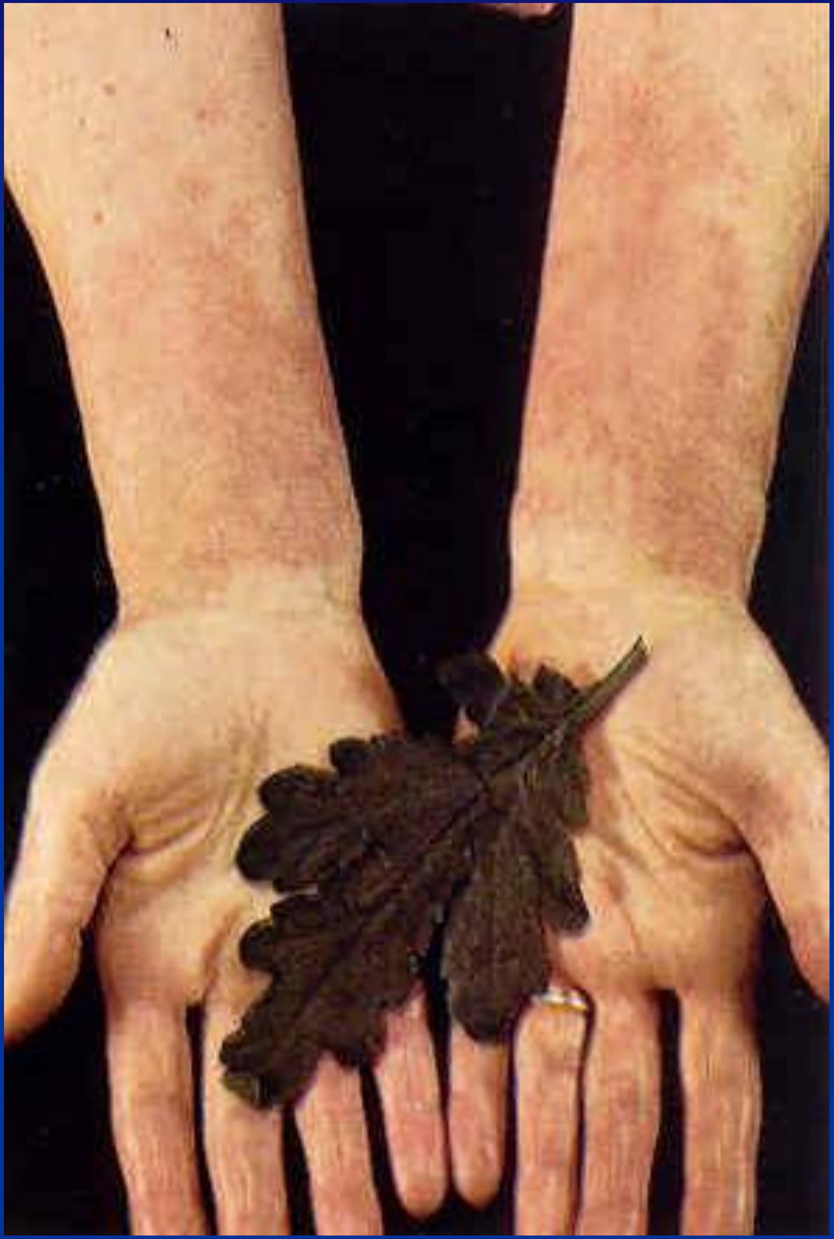
Linear distribution with satellite lesions



What do you think?

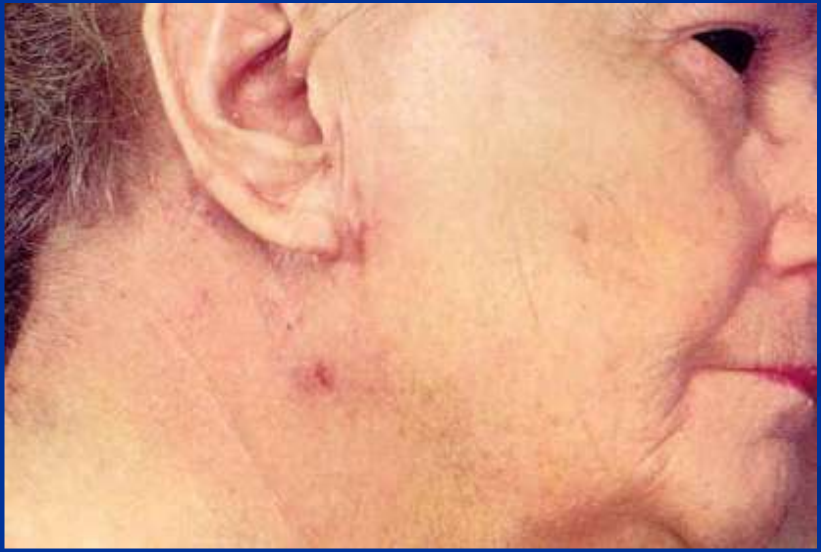


Bilateral
but…..



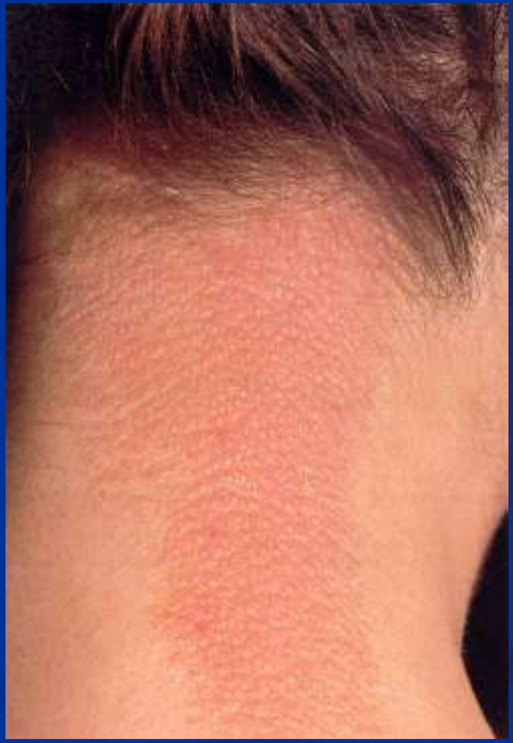
Subacute Allergic Eczema

- Note slight swelling and erythema
- No lichenification
- Location – what could be the cause?



Chronic Allergic Eczema

- Note the hyperkeratosis, lichenification and fissuring



Toxic / Irritant Eczema

(occurring in non allergic skin)

- Characteristics:
- Accounts for 75% of exogenous eczema
- Age, race and sex are insignificant
- Results from repeated exposure to toxic or subtoxic agents
- Severity of skin symptoms vary with the individual and the type of irritant and the length of contact
- Includes sx of itching, stinging and burning
- Usually associated with chronic disturbance of the barrier function of the skin

Toxic/Irritant Eczema cont.

Common causes:

- Repeated exposure to alkaline detergents
- Repeated exposure to organic solvents
- Corrosive agents
- Industrial chemicals
- Chronic self perpetuating habits that irritate the skin

Toxic/Irritant Eczema cont.

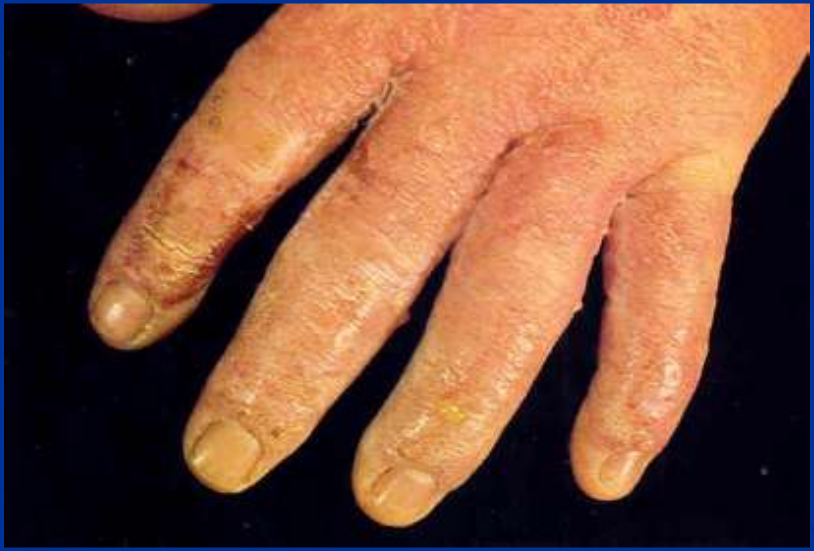
Treatment:

- Remove the cause
- Application of emollients
- Use of soap substitutes
- Barrier creams
- Borrow’s or potassium permanganate soaks twice daily

Biopsy/testing- usually not necessary

Acute Toxic/Irritant Eczema

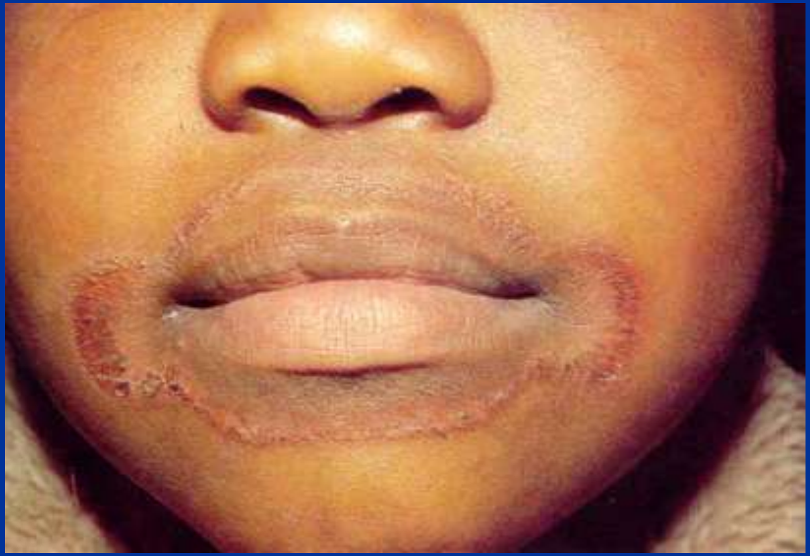
Note: distribution, swelling and weeping



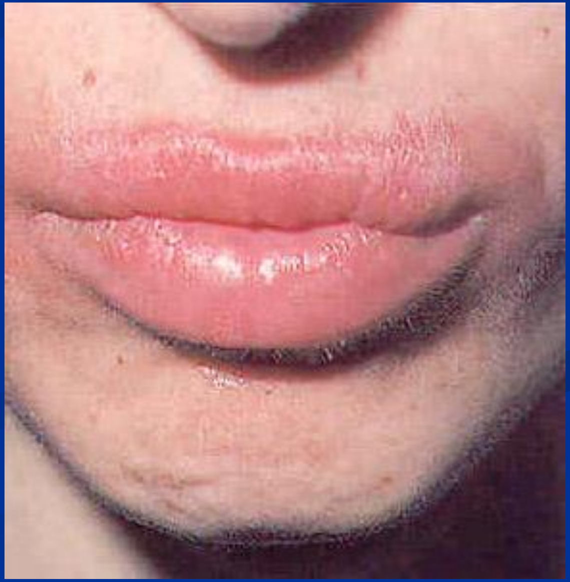
Subacute Toxic/Irritant Eczema

Lip licking

- often seen in children who have atopic eczema
- Variant of irritant eczema

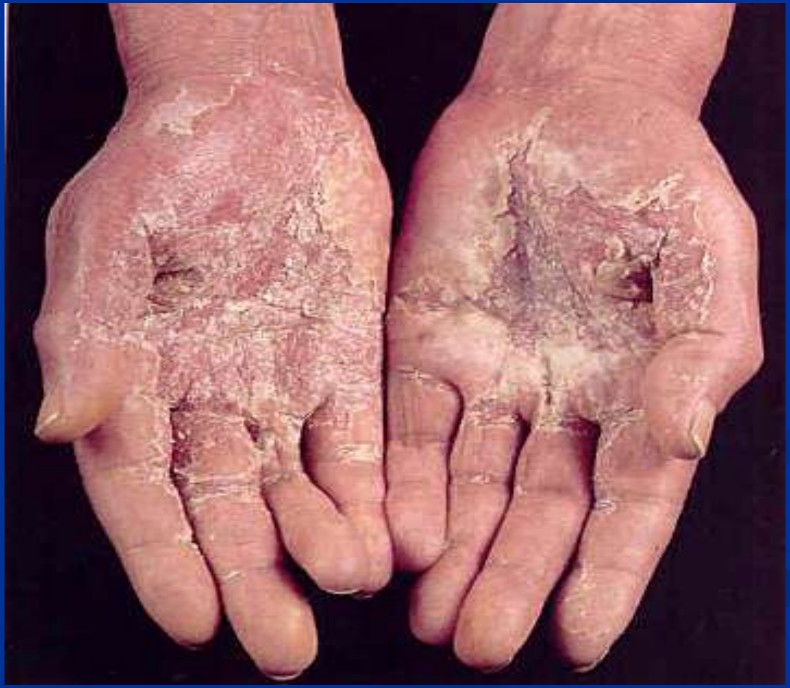


compare



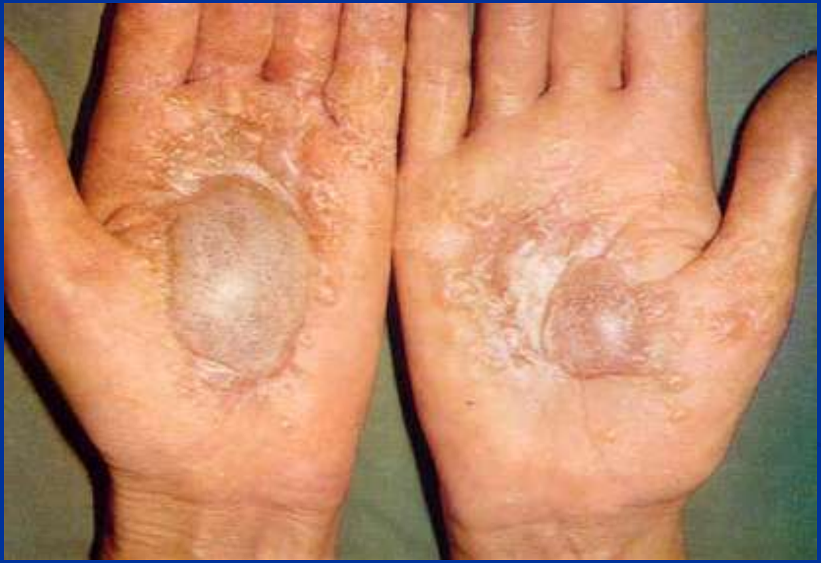
Chronic Toxic/Irritant Eczema

- Note:papulosquamous dermatosis with hyperkeratosis, maceration, fissuring and erosions
- Eruptions tend to be sore rather than itching



Acute, subacute, or chronic?

Swelling? Erythema? Desquamation? Lichenification?



Comparison of Classifications of the 3 common types of eczema

ACUTE	**Atopic IgE**	**Toxic/ Irritant**	**Allergic Contact**
Erythema	X	X	X
Papules	X	NA	X
Vesicles	X	X	X
Erosions	X	X	X
Crusts	X	X	X
Scales	X	NA	X
Sharp/ confined	NA	X	X
Spreading peripherally	Flexor surfaces, neck, eye lids, d foot	X	NA
Onset rapid	Before age 12	X	Usually as adult
Onset slow	NA	NA	X
Concentration L/H	NA	H	L
Incidence	Other signs	Anyone	Sensitized

Comparison of Classifications

Chronic	**Atopic** **IgE**	**Toxic/Irritant**	**Allergic/Contact**
scaling	X	X	X
fissues	X	X	X
crusts	NA	X	X
papules	NA	X	X
excoriation	X	NA	X
lichenification	X	NA	X
Periorbital pigmentation	X	NA	NA
Infraorbital folds in the eyelids	X	NA	NA
Foillicular papules	X more common in the black pop.	NA	NA

Pompholyx (from Greek word meaning blister)

- Characteristics:
- Intense itching and burning proceed lesions
- Blisters and vesicles on hands/ feet
- Becomes highly exudative
- Dries up in about 2 wks leaving painful fissuring
- Acute symptom of a chronic problem
- Usually no cause but can be due to trichophytin and associated with fungal infection of the feet

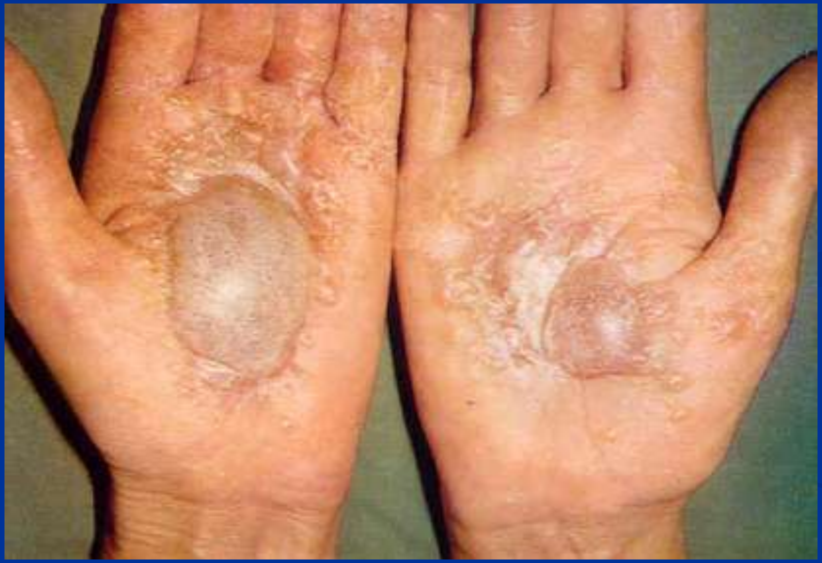
Pomhpolyx cont.

- Treatment:
- Avoidance of soap
- Emollients
- Soaks (burrows or potassium permanganate)
- Potent or very potent topical steroids with or with occlusion
- Antibiotics for infection
- Systemic steroids
- Coal tar extracts
- Biopsy/testing- usually not necessary

Pompholyx

Where else should you look?

What else might this be call?



Nummular Eczema

- **Characteristics:**
- **usually -**personal or family history of allergy, especially asthma, hay fever, and childhood eczema

- **Distinctive Characteristics -** Coin-shaped papulovesicular patches that develop in to scaling and crusting lesions; lesions may be as large as 4-5cm in diameter with distinct margins, initial eruptions on arms and legs; intense itching; tends to be chronic

Nummular Eczema cont.

- **Characteristics:**
- Most severe during winter; may be aggravated by systematic administration of iodine or bromine; secondary bacterial infections are common

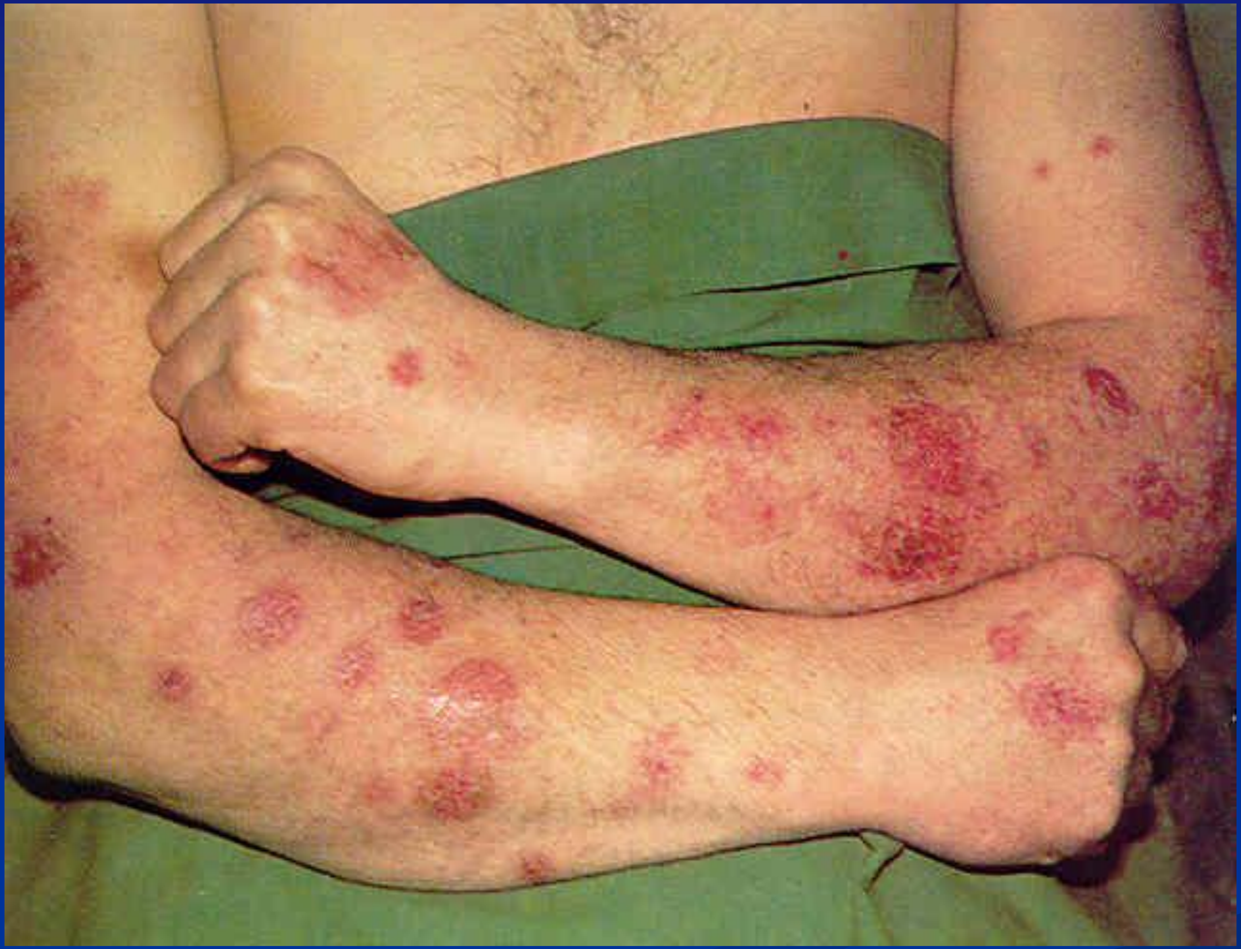
- **Treatment:** skin hydration, topical corticosteroids, intralesional injection, coal tar ointments, UVB treatment, treat secondary infection

Nummular Eczema cont.

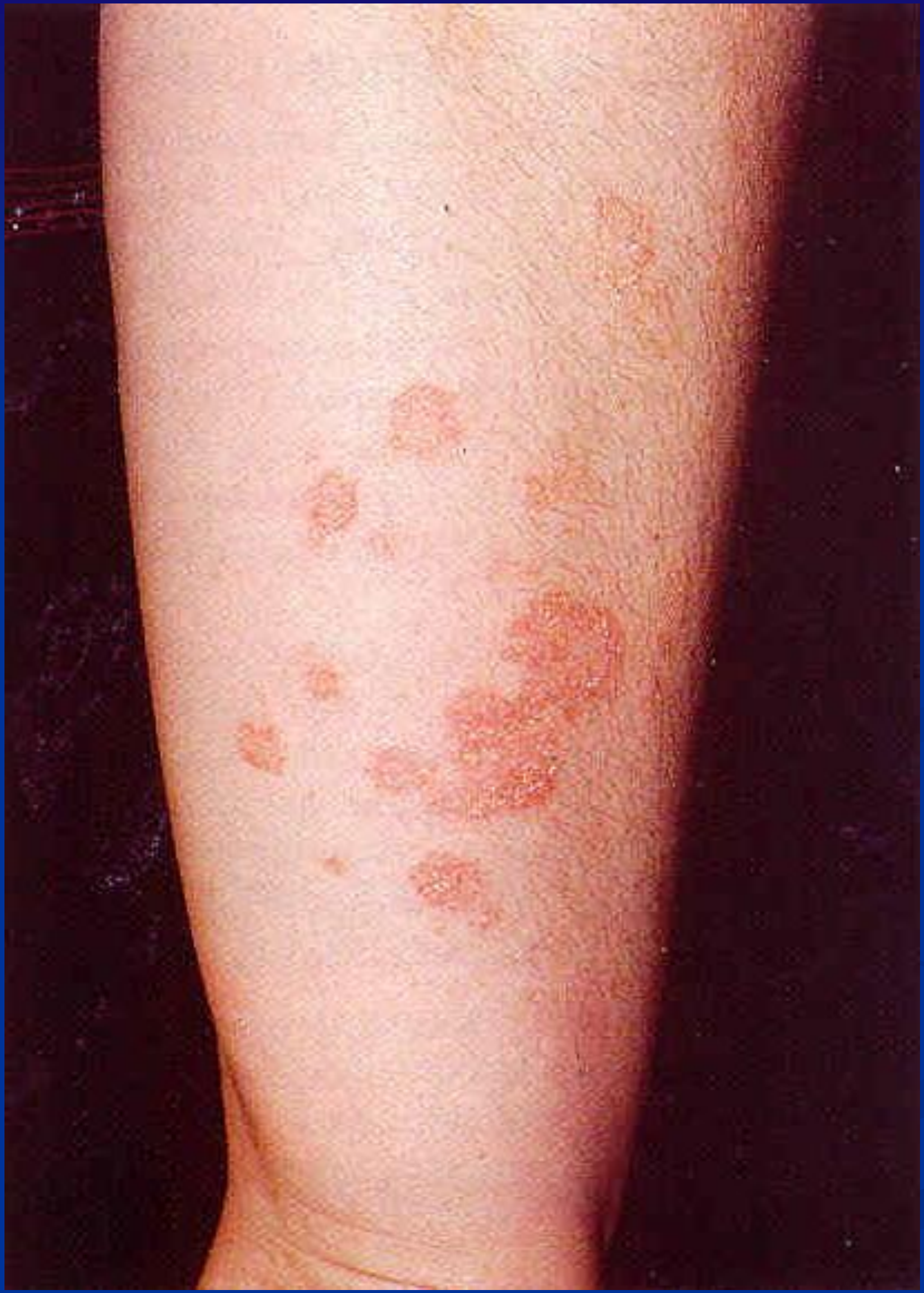
- **Confused with** – contact dermatitis/eczema, atopic eczema, psoriasis, impetigo, tinea corporis
- **Biopsy/testing –** not usually necessary

Note:

- Coin shaped lesions
- dorsal surface arms
- bilateral



What else should you think about?



Seborrehea

- **Characteristics:** Positive family history is common
- Seen in all age groups equally
- May occur on presternal area and mid upper back
- Stress may increase symptoms
- Pityrosporum ovale may be causative factor

- **Distinctive Characteristics:**
- Red greasy scaling rash consists of patches and plaques with indistinct margins and an underlying red glazed look to the skin
- Most commonly located in the hairy areas, nasolabial folds, retroauriclar folds
- Excoriations from scratching are rare

Seborrhea cont.

Treatment:

Scalp –

- try OTC preps first (antidandruff, tar or ketoconazole shampoo)
- Steroid lotions for very short term use
- 10% Liquor Carbonis Detergens HS and shampoo in AM with Dawn Detergent

Skin -

- try OTC’s first
- corticosteroids (mild to moderate potency) and/or ketoconazole topically

Seborrhea cont.

Eye lashes-

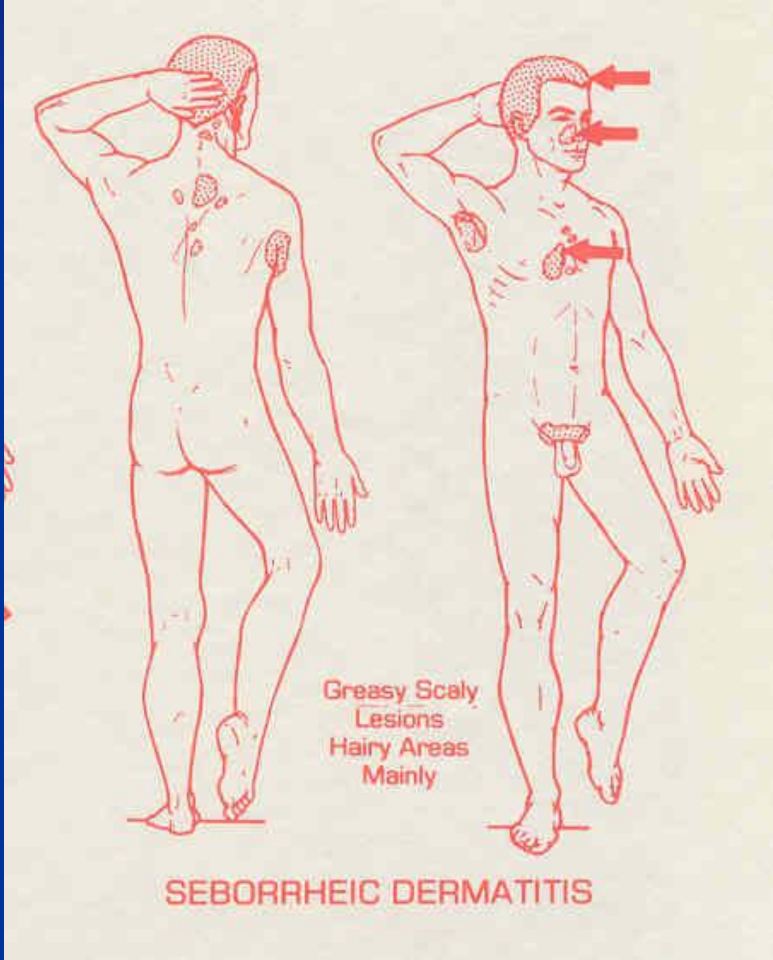
- Warm compresses and gentle debridment with Q tip
- Sulfacetamide ophthalmic ointment applied topically

** Areas that become exudative may be treated with potassium permanganate or burrow’s soaks

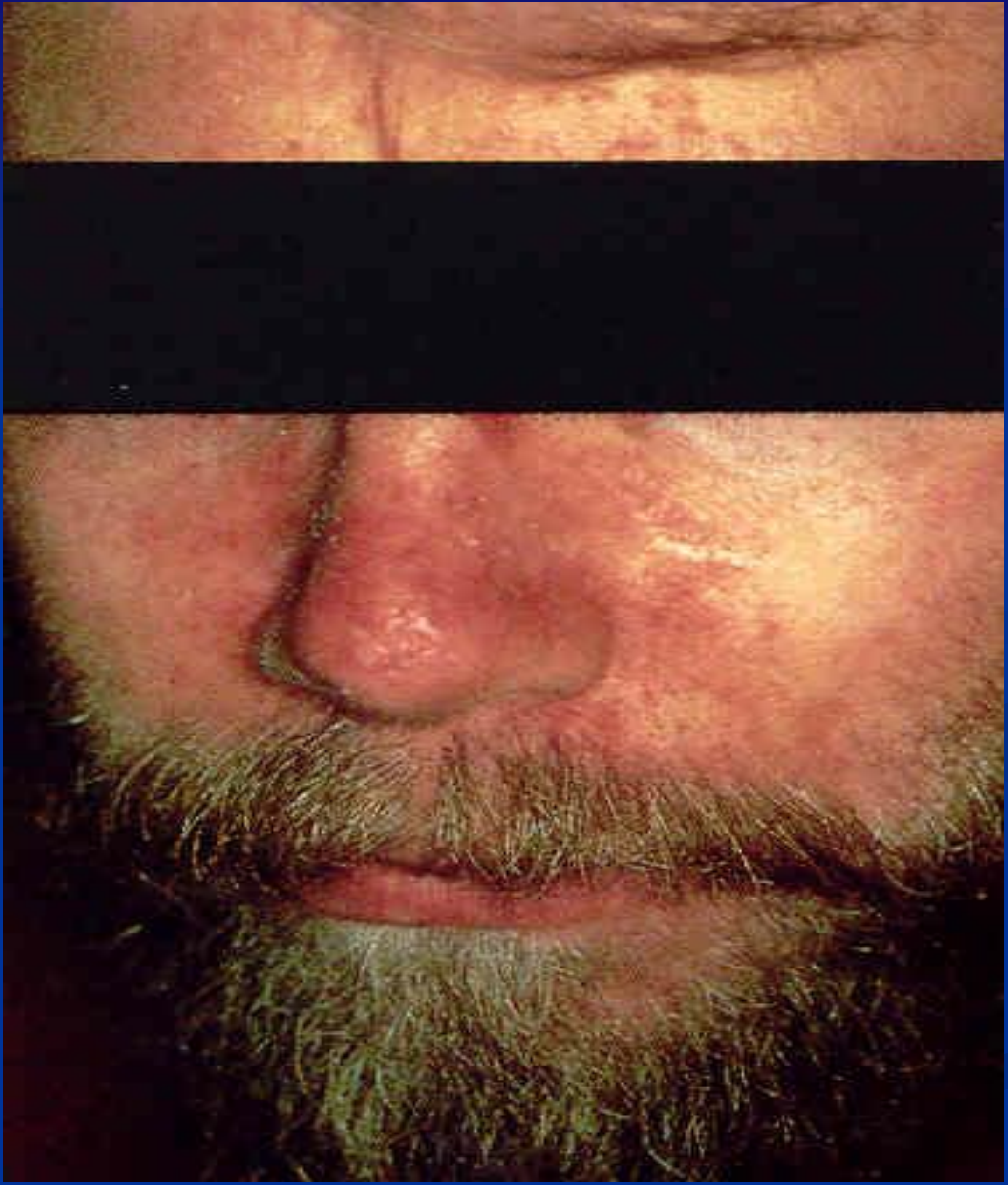
Confused with – atopic dermatitis, eczema, psoriasis, discoid lupus, tinea

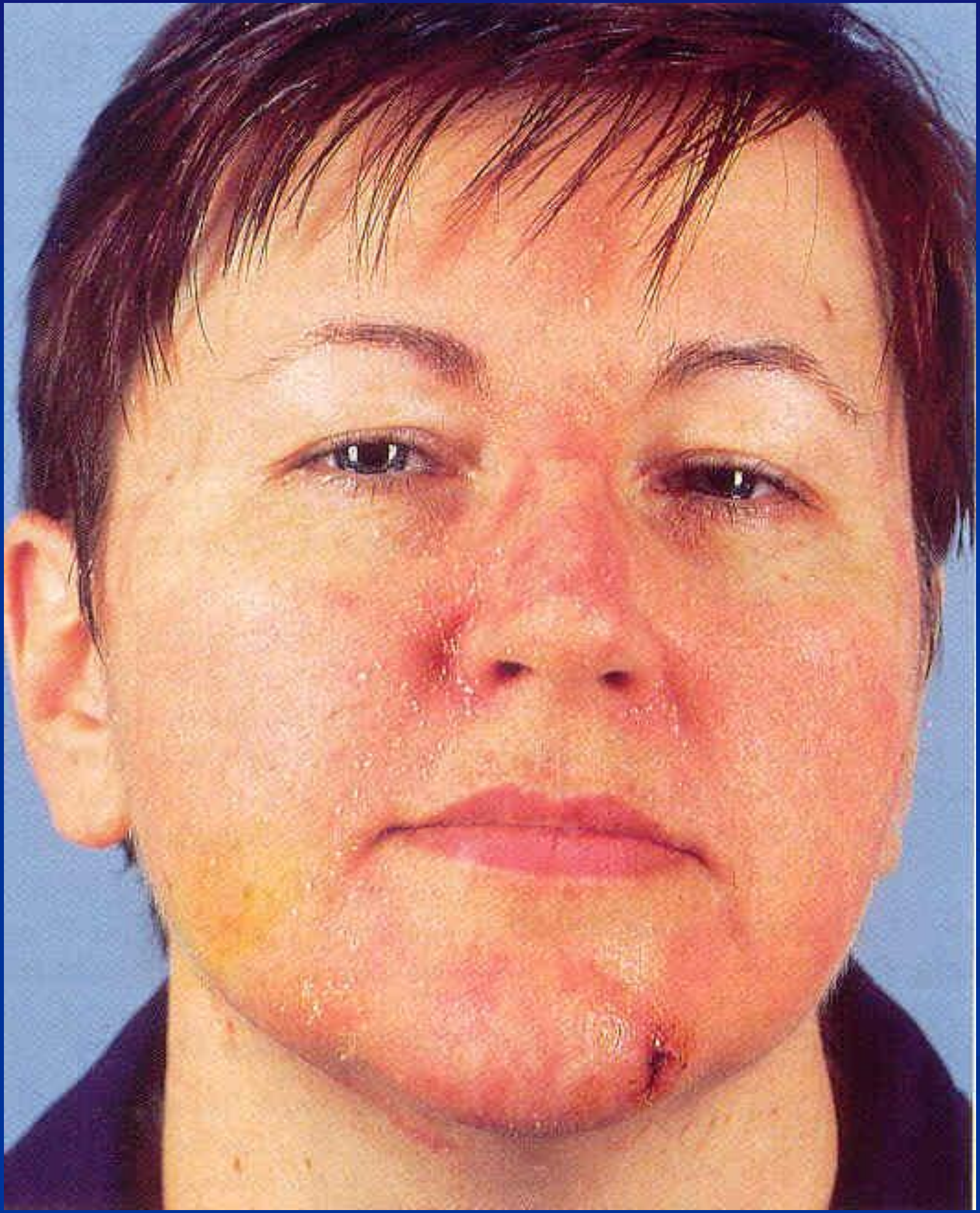
Biopsy/testing – usually none

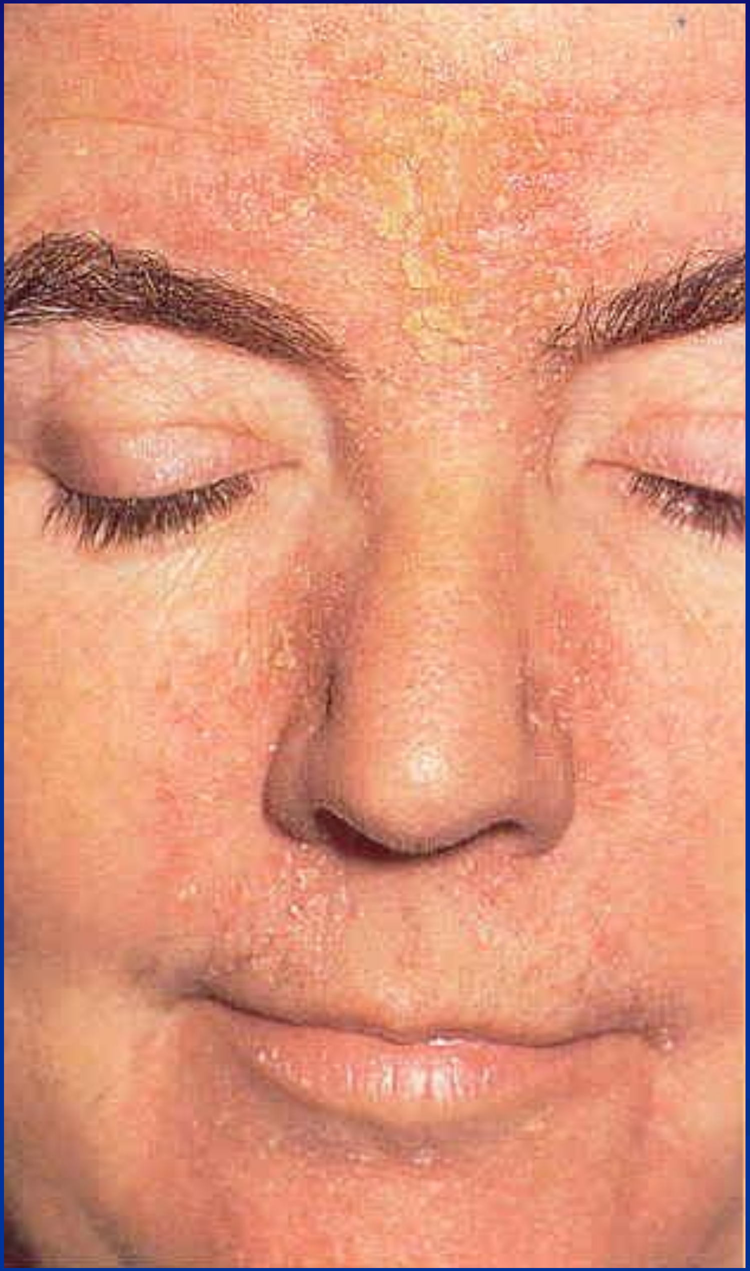
Seborrhea Distribution


Greasy Scaly
Lesions
Hairy Areas
Mainly
SEBORRHEIC DERMATITIS

What else could this be?







Asteatotic Eczema
(Xerotic Eczema, “Winter Itch”)

Characteristics:

- Seen mainly in elderly
- Worse in the winter
- Precipitated by excessive washing

Treatment:

- Avoid excessive washing and use of soap
- Emollients
- Increase humidity in the environment
- Topical steroids for a short periods of time

Localized Neurodermatitis Cont.

- **Treatment:**
- Stop the scratching
- Occlusive steroid dressings esp. at night
- Lubrication
- Doxepin ointment and/or po 10-20mg
- Hydroxyzine at night
- Intralesional steroid injection
- Stress management and/or medication
- Treatment is longterm and may be unsatisfactory
- **Confused with** – atopic eczema, psorasis, tinea, seborrhea
- **Biopsy / testing** – none usually necessary

Localized Neurodermatitis

(known as Lichen Chronicus Simplex)

- **Characterisitcs:**
- Origin often small patch of dermatitis or insect bite starting the itch –scratch- itch cycle
- Condition unrelated to allergies or family history
- More common in women
- Nonseasonal
- aggravated by stress
- worse at night
- may be secondary to atopic eczema, contact dermatitis, lichen planus, psoriasis, or insect bite

Localized Neurodermatitis

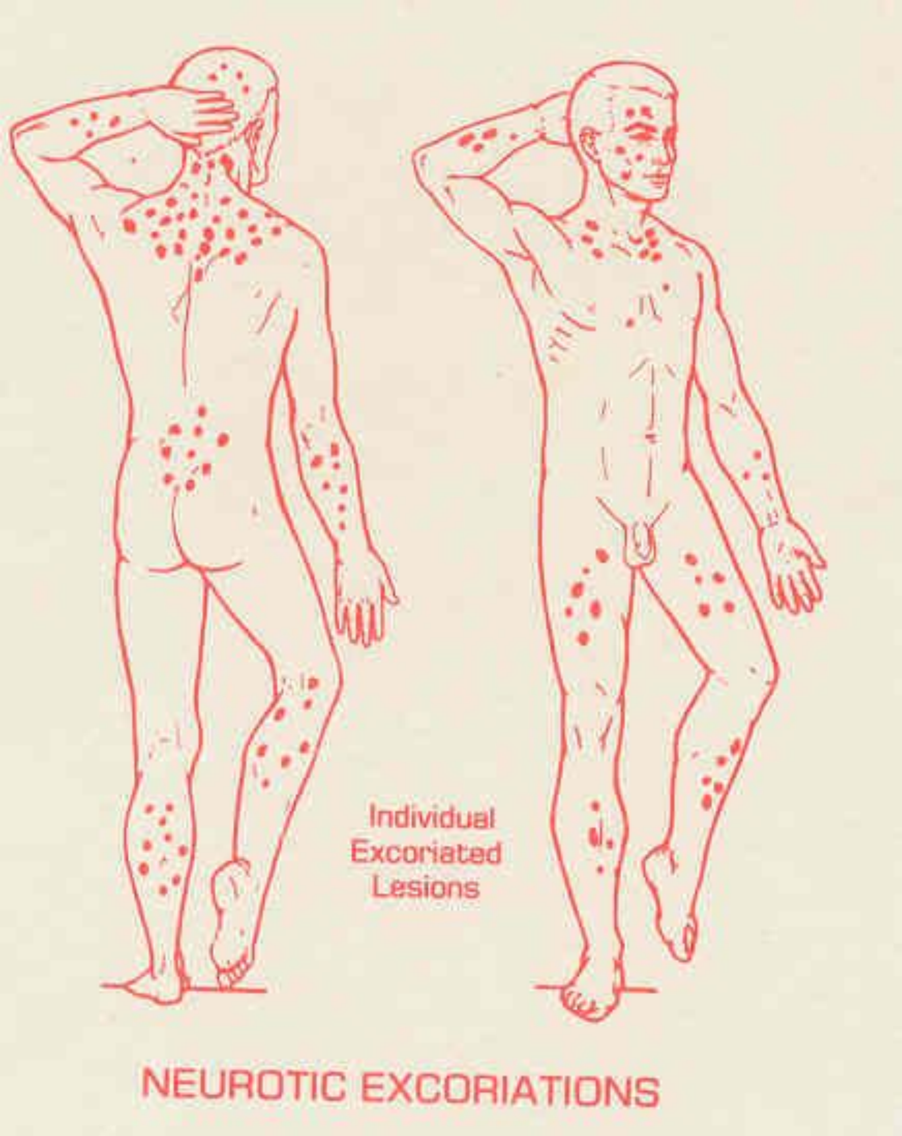
(known as Lichen Chronicus Simplex) CONT.

Distinctive Characteristics:

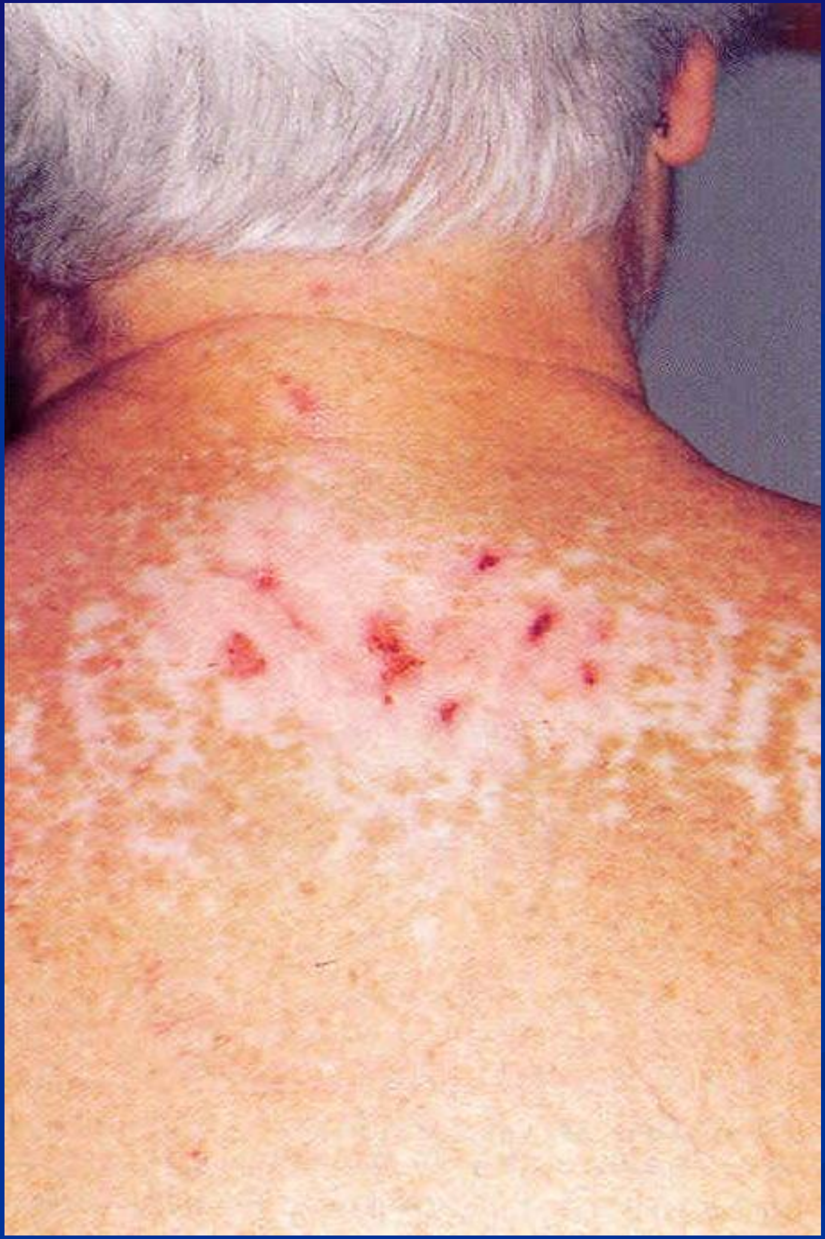
- Lesions lichenified or excoriated
- usually limited to a single patch at hairline of nape of neck or on wrists, ankles, ears, or anal area
- Not bilateral
- Llichenification of dark skin develops a “follicular pattern”

Localized Neurodermatitis Distribution

(known as Lichen Chronicus Simplex)


Individual
Excoriated
Lesions
NEUROTIC EXCORIATIONS

What should you think about in this man?



Summary of Treatment

1. **Conservative Therapy**
    a) Education (chronicity, prevention, and trigger id)
    b) Use of astringents and emollients/moisturizers
    c) OTC products (hydrocortisone, Benadryl, Calamine, etc.)
2. **Low to mid potency steroid creams**
3. **High potency steroid creams**
4. **Immunomodulators -** Elidel and Protopic creams
5. **Nontraditional agents**
6. **PO therapy:** antiprurutics, steroids, cyclosporine, methotrexate
7. **Coal Tar**
8. **PUVA therapy** (phototherapy)

Drug eruptions

Drug eruptions

- Almost any drug can cause a cutaneous reaction
- Many inflammatory skin conditions can be caused or exacerbated by drugs

Mechanisms

1. Non-allergic drug reactions

- result of overdosage, accumulation of drugs, unwanted pharmaco-logical effects, idiosyncratic, or a result of alterations of ecological balance
- They are a normal biological effect
- often predictable
- affect many, or even all, patients taking the drug at a sufficient dosage for a sufficient time

Mechanisms

2. Allergic drug reactions

- less predictable.
- occur in only a minority of patients receiving a drug
- occur even with low doses
- They are not a normal biological effect of the drug and usually appear after the latent period required for induction of an immune response
- Chemically related drugs may cross-react
- majority of allergic drug reactions are caused by cell-mediated immune reaction

Some mechanisms involved in drug reactions

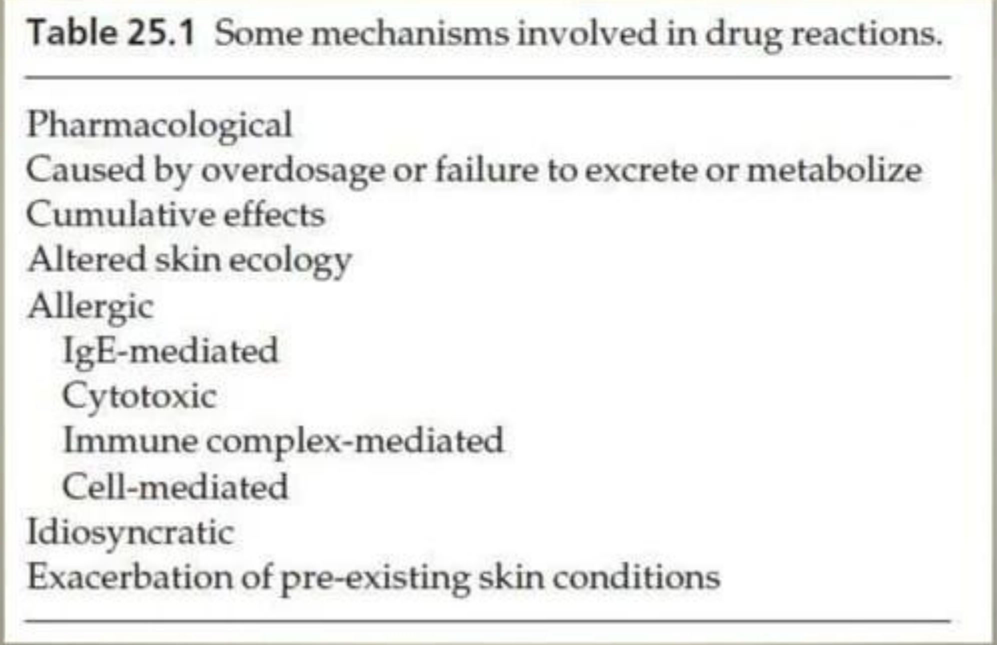


Table 25.1 Some mechanisms involved in drug reactions.

- Pharmacological
- Caused by overdosage or failure to excrete or metabolize
- Cumulative effects
- Altered skin ecology
- Allergic
  - IgE-mediated
  - Cytotoxic
  - Immune complex-mediated
  - Cell-mediated
- Idiosyncratic
- Exacerbation of pre-existing skin conditions

Presentation

Antibiotics

Penicillins and sulphonamides

- are most commonly causing allergic reactions.
- These are often morbilliform but urticaria, erythema multiforme and fixed eruptions are common too.
- DDx. Is viral infections as often associated with exanthems
- Most patients with infectious mononucleosis develop a morbilliform rash if ampicillin is administered.
- Penicillin is a common cause of severe **anaphylactic reactions**, which can be life-threatening.

Minocycline

- can accumulate in the tissues and produce a brown or grey colour in the mucosa, sun-exposed areas or at sites of inflammation, as in the lesions of acne
- hepatitis, worsen lupus erythematosus or elicit a transient lupus-like syndrome

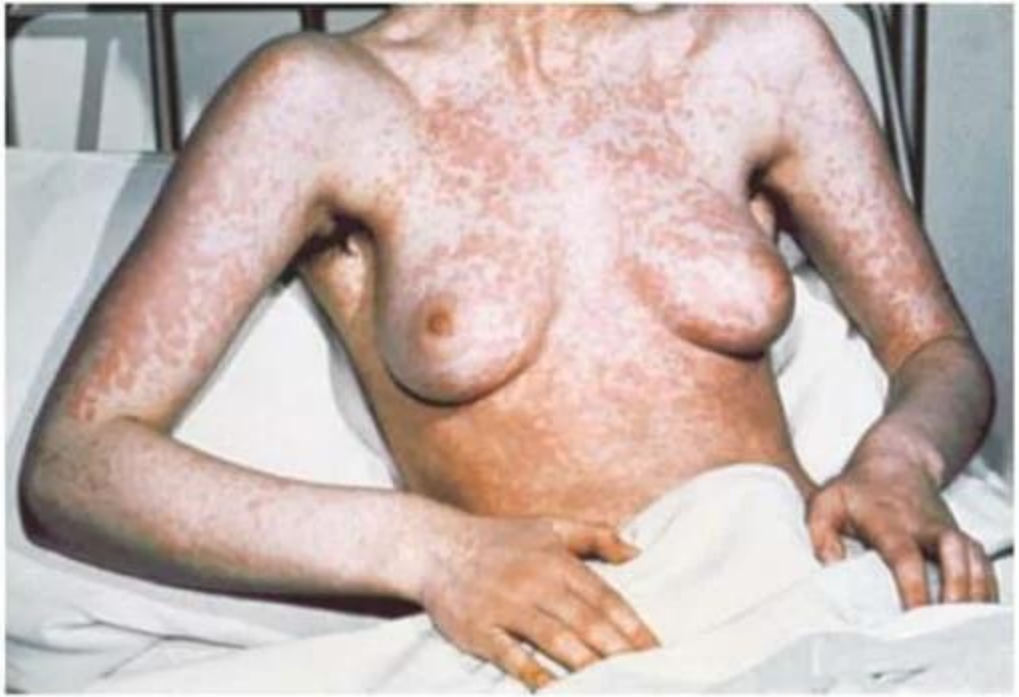


Fig. 25.2 Symmetrical erythematous maculopapular rash as a result of ampicillin.

Oral contraceptives

- Reactions to these are less common now that their hormonal content is small.

1. telogen effluvium
2. Melasma, hirsutism
3. erythema nodosum
4. acne
5. photosensitivity

Steroids

Cutaneous side-effects from systemic steroids include:

- a ruddy face
- cutaneous atrophy
- striae
- hirsutism
- an acneiform eruption
- a susceptibility to cutaneous infections, which may be atypical

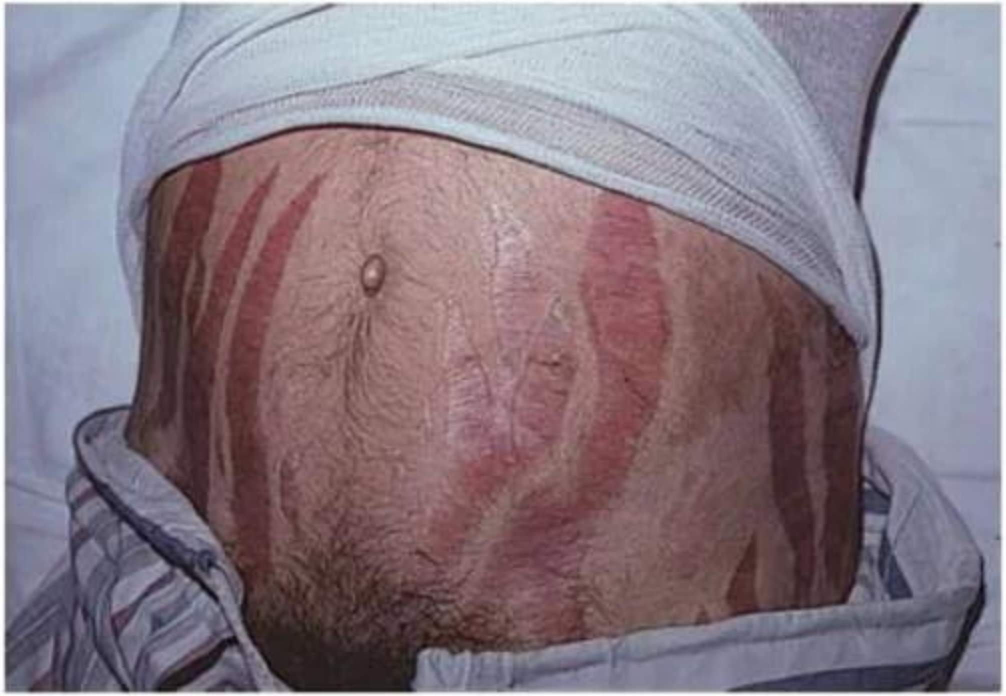


Fig. 25.1 Gross striae caused by systemic steroids.

Anticonvulsants

Skin reactions to phenytoin, carbamazepine, lamotrigine and phenobarbital are common and include:

- erythematous, morbilliform, urticarial and purpuric rashes.
- rarely TEN, erythema multiforme, exfoliative dermatitis, DRESS syndrome and a lupus erythematosuslike syndrome

Some common reaction patterns and drugs that can cause them

Toxic (reactive) erythema

- most common type of drug eruption
- looking like measles or scarlet fever, and sometimes showing prominent urticarial or erythema multiforme-like elements.
- Itching and fever may accompany the rash.
- Culprits include antibiotics (especially ampicillin), sulphonamides and related compounds (diuretics and hypoglycaemics) and barbiturates

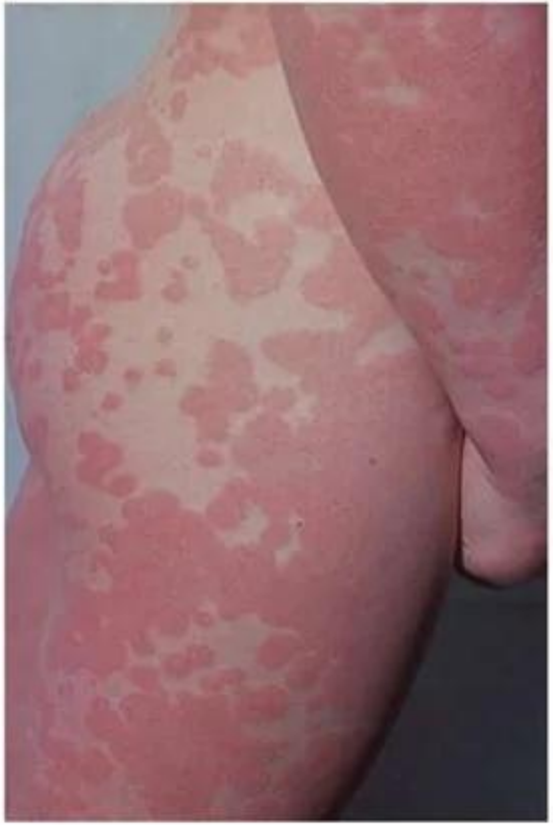


Fig. 25.4 Toxic erythema with urticarial features.

Urticaria

- salicylates are the most common, often working non-immunologically as histamine releasers.
- Antibiotics
- Urticaria may be part of a severe and generalized reaction (anaphylaxis) that includes bronchospasm and collapse

Erythema multiforme and Stevens–Johnson syndrome

- Sulphonamides, barbiturates, lamotrigine and phenylbutazone

Purpura

- Thiazides, sulphonamides, barbiturates and anticoagulants

Bullous eruptions

- also in Stevens–Johnson syndrome
- Vancomycin, lithium, diclofenac, captopril, furosemide and amiodarone are associated with development of linear IgA bullous disease

Fixed drug eruptions

- Round erythematous or purple, and sometimes bullous plaques recur at the same site each time the drug is taken
- Pigmentation persists between acute episodes.
- The glans penis seems to be a favoured site.
- The causes of fixed drug eruptions in any country follow the local patterns of drug usage
- **Paracetamol** is currently the most common offender in the UK
- **Trimethoprim-sulfa** leads the list in the USA
- NSAIDs (including aspirin), antibiotics, systemic antifungal agents and psychotropic drugs lie high on the list of other possible offenders.

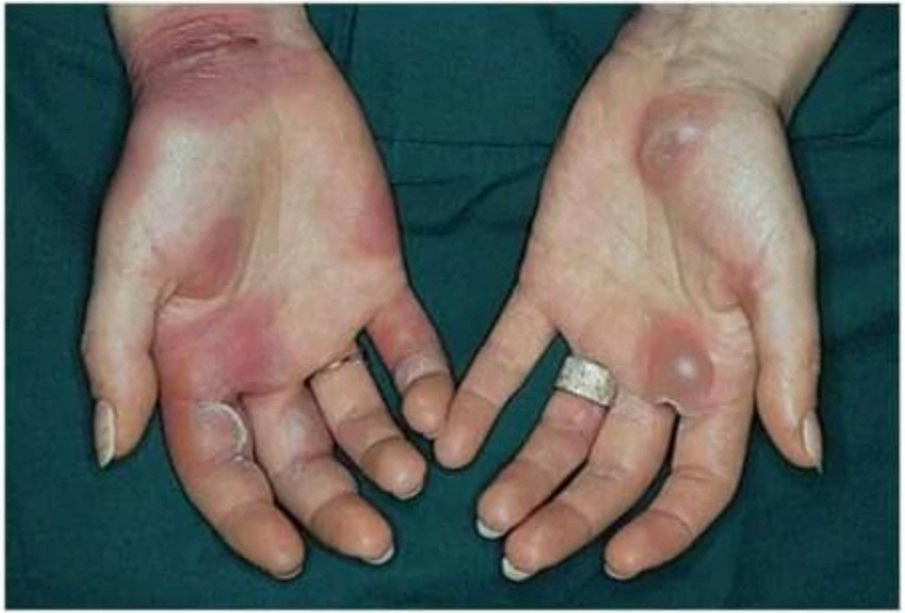


Fig. 25.6 Fixed drug eruption – an unusually severe bullous reaction.

Acneiform eruptions

- Lithium, iodides, bromides
- oral contraceptives, androgens or glucocorticosteroids
- Antitub erculosis and anticonvulsant

Lichenoid eruptions

- These resemble lichen planus but mouth lesions are uncommon and scaling and eczematous elements may be seen.
- antimalarials, NSAIDs, gold, phenothiazines

Hair loss

- Retinoid
- cytotoxic agents
- oral contraceptive

Hypertrichosis

- dose-dependent effect of diazoxide, minoxidil and ciclosporin

Pigmentation

- Melosma in oral contraceptive plus sun exposure
- Large doses of phenothiazines impart a blue–grey colour to exposed areas
- clofazimine makes the skin red
- mepacrine turns the skin yellow
- minocycline turns areas of leg skin a curious greenish grey colour

Xerosis

- oral retinoids
- nicotinic acid
- lithium

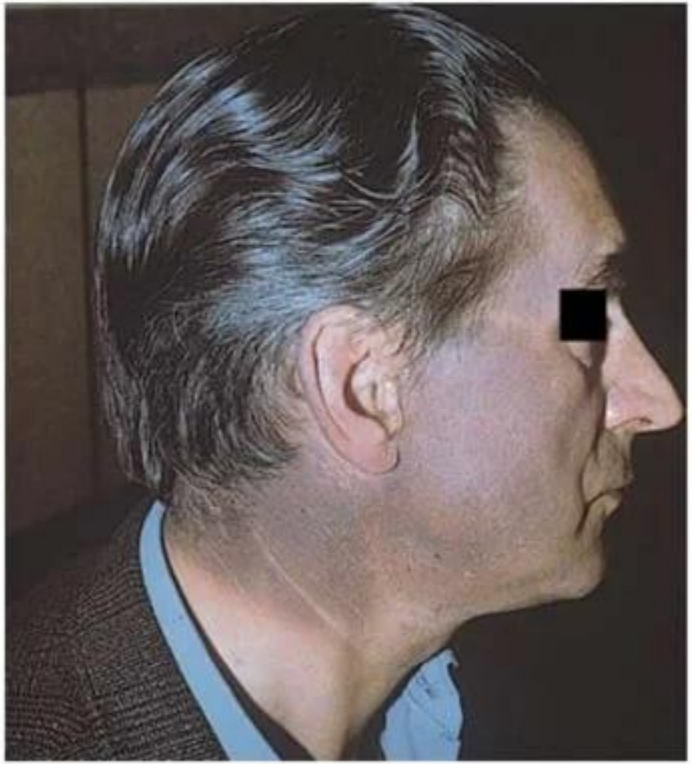


Fig. 25.8 Note sparing of skin creases and area shielded by spectacle frames in this patient with photorelated hyperpigmentation from a phenothiazine drug.

Course

- If an allergic reaction occurs during the first course of treatment, it characteristically begins late, often about the ninth day, or even after the drug has been stopped
- In previously exposed patients the common morbilliform allergic reaction starts 2–3 days after the administration of the drug
- The speed with which a drug eruption clears depends on the type of reaction and the rapidity with which the drug is eliminated

Differential diagnosis

- Ranges over the whole subject of dermatology
- The general rule is never to forget the possibility of a drug eruption when an atypical rash is seen. Six vital questions should be asked

The six vital questions to be asked when a drug eruption is suspected.

1. Can you exclude a simple dermatosis (e.g. scabies or psoriasis) and the known skin manifestations of an underlying disorder (e.g. systemic lupus erythematosus)?
2. Does the rash itself suggest a drug eruption (e.g. urticaria, erythema multiforme)?
3. Does a past history of drug reactions correlate with current prescriptions?
4. Was any drug introduced a few days or weeks before the eruption appeared?
5. Which of the current drugs most commonly cause drug eruptions (e.g. penicillins, sulphonamides, thiazides, allopurinol, phenylbutazone)?
6. Does the eruption fit with a well-recognized pattern caused by one of the current drugs (e.g. an acneiform rash from lithium)?

Treatment

- The first approach is to withdraw the suspected drug, accepting that several drugs may need to be stopped at the same time.
- The decision to stop or continue a drug depends upon:

1. the nature of the drug
2. the necessity of using the drug for treatment
3. the availability of chemically unrelated alternatives
4. the severity of the reaction, its potential reversibility
5. the probability that the drug is actually causing the reaction.

- Every effort must be made to correlate the onset of the rash with prescription records.
- Often, but not always, the **latest drug** to be introduced is the most likely culprit.
- **Prick tests** and *in vitro* tests for allergy are unreliable to
- **Re-administration**, as a diagnostic test, is usually unwise except when no suitable alternative drug exists

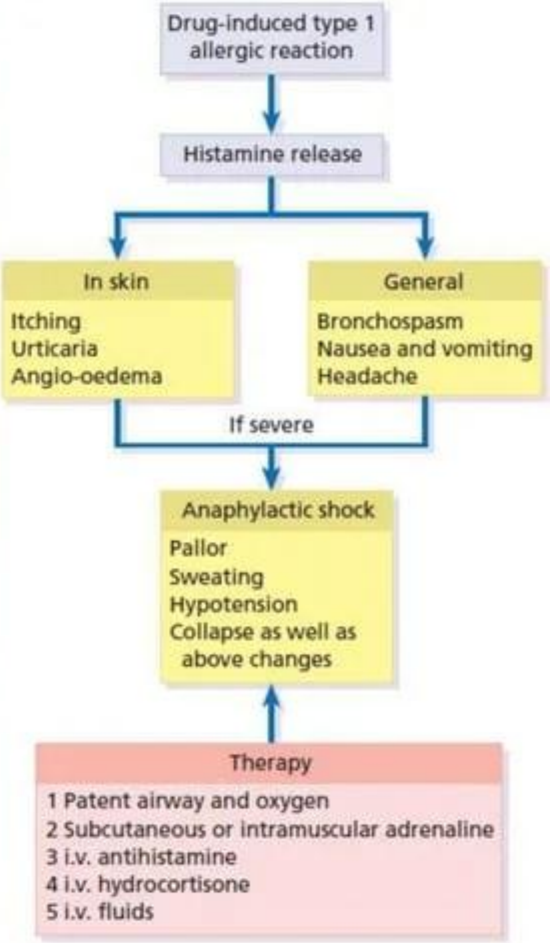
Non-specific therapy depends upon the type of eruption.

- In urticaria, antihistamines are helpful.
- In some reactions, topical or systemic corticosteroids can be used, and applications of calamine lotion may be soothing.
- Plasmapheresis and dialysis can be considered in certain life-threatening situations

Anaphylactic reactions

- ensure that the **airway** is not compromised (e.g. oxygen, assisted respiration or even emergency tracheostomy).
- **One or more injections of adrenaline** (epinephrine) (1 : 1000) 0.3–0.5 mL should be given subcutaneously or intramuscularly in adults
- slow (over 1 min) **intravenous injection of chlorphenamine maleate** (10–20 mg diluted in syringe with 5–10 mL blood).
- Although the action of intravenous **hydrocortisone** (100 mg) is delayed for several hours, it should be given to prevent further deterioration in severely affected patients.
- Patients should be observed for 6 h after their condition is stable, as late deterioration may occur.
- If an anaphylactic reaction is anticipated, patients should be taught how to self-inject adrenaline, and may be given a salbutamol inhaler to use at the first sign of the reaction

The cause, clinical features and treatment of anaphylaxis.



Drug-induced type 1 allergic reaction

Histamine release

In skin

- Itching
- Urticaria
- Angio-oedema

General

- Bronchospasm
- Nausea and vomiting
- Headache

If severe

Anaphylactic shock

- Pallor
- Sweating
- Hypotension
- Collapse as well as above changes

Therapy

1 Patent airway and oxygen
2 Subcutaneous or intramuscular adrenaline
3 i.v. antihistamine
4 i.v. hydrocortisone
5 i.v. fluids

Desensitization

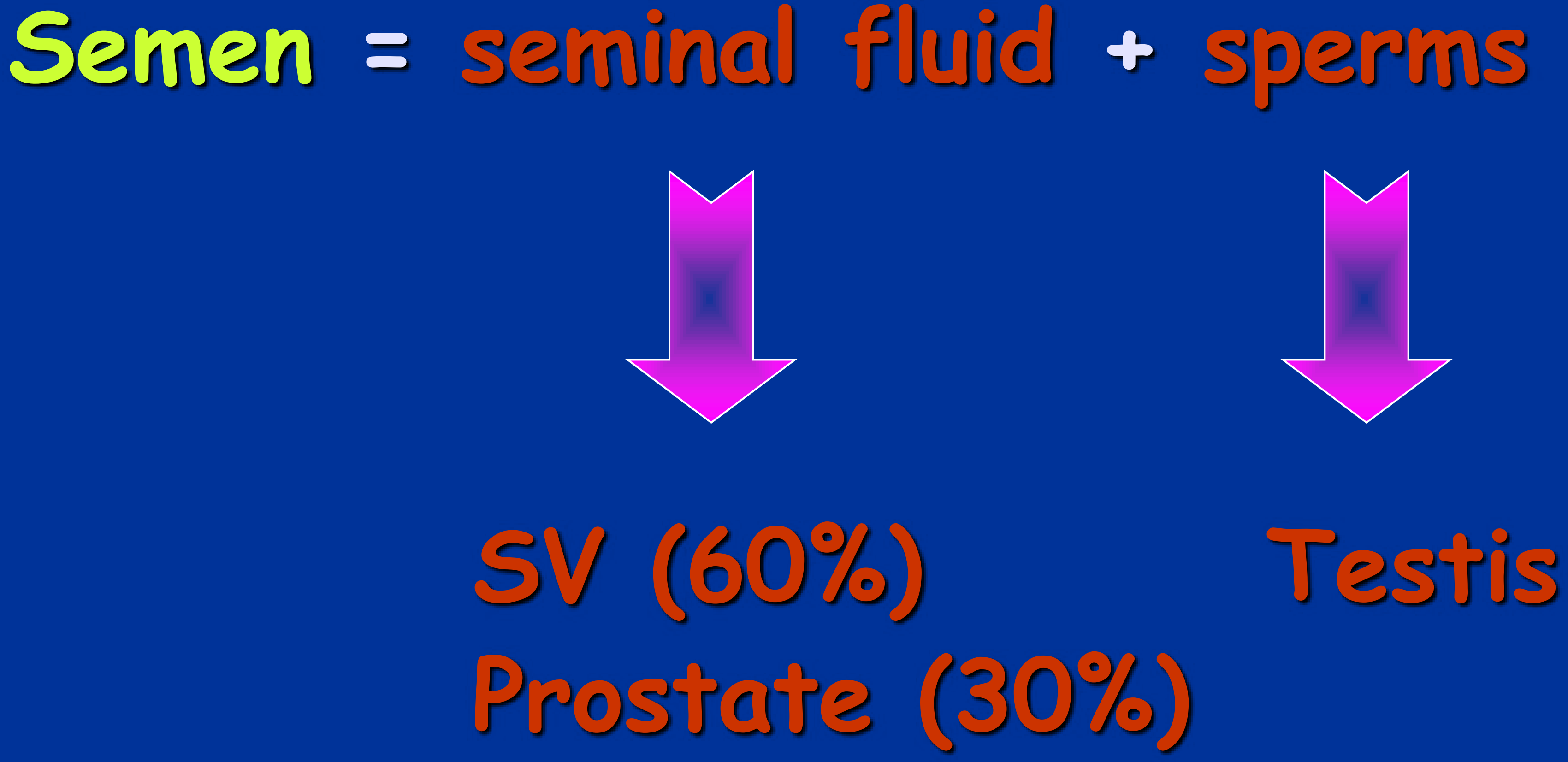
- is seldom advisable or practical

The End

بسم الله الرحمن الرحيم

Male Infertility

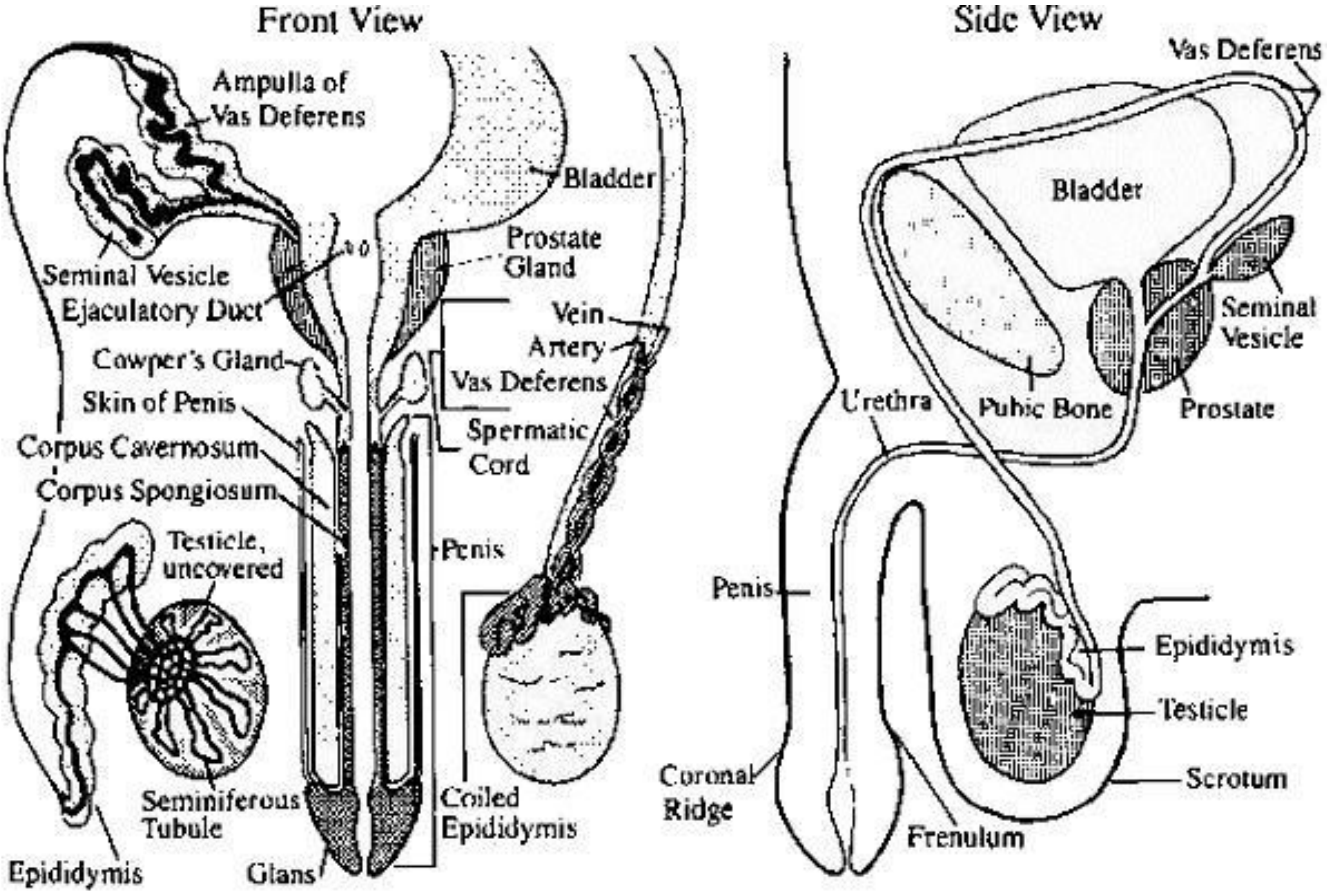
By
Dr. Mohammad M. Abdulgabbar
Assistant Professor of Andrology & STDs
Sana'a University



Semen = seminal fluid + sperms

SV (60%)
Prostate (30%)

Testis

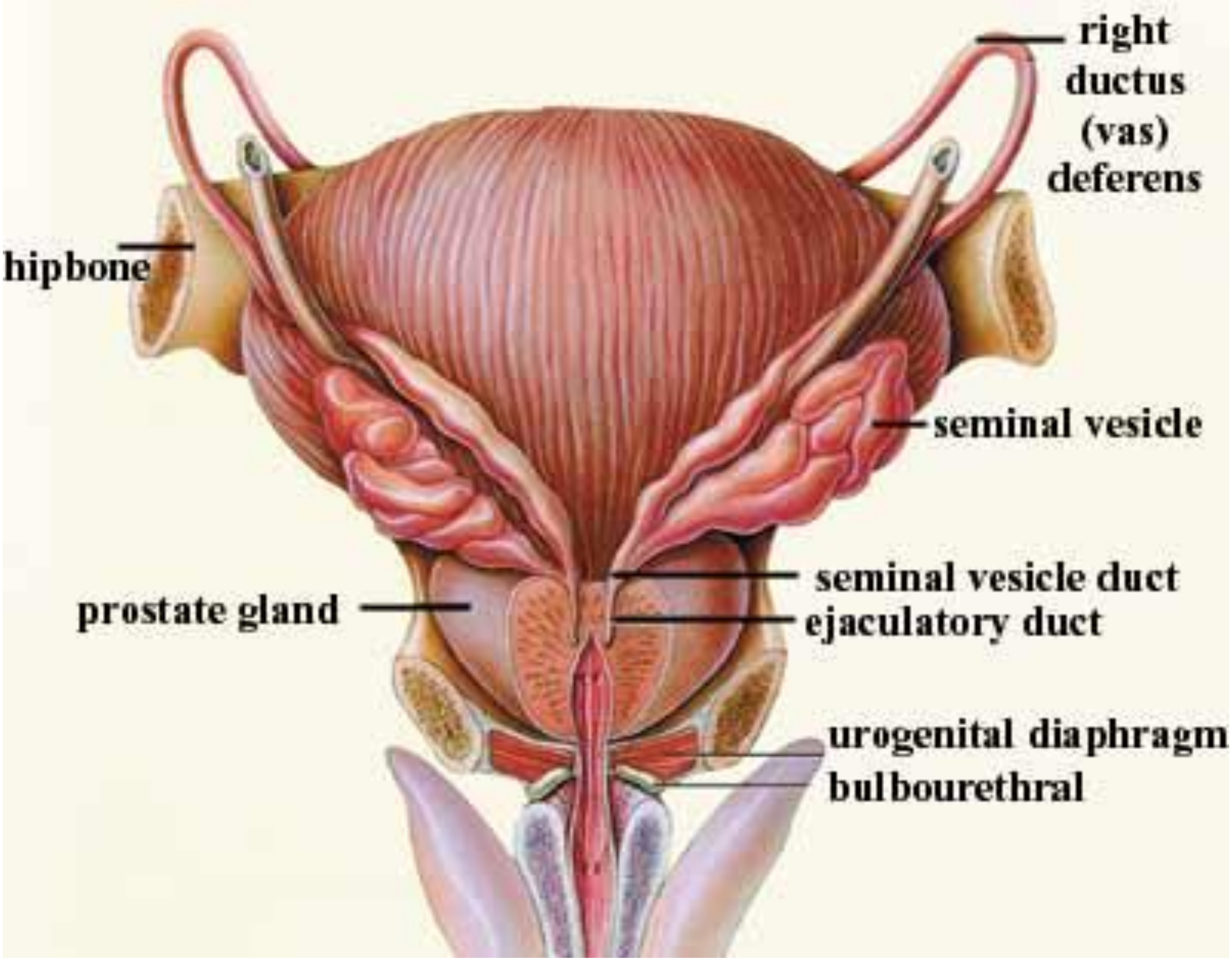


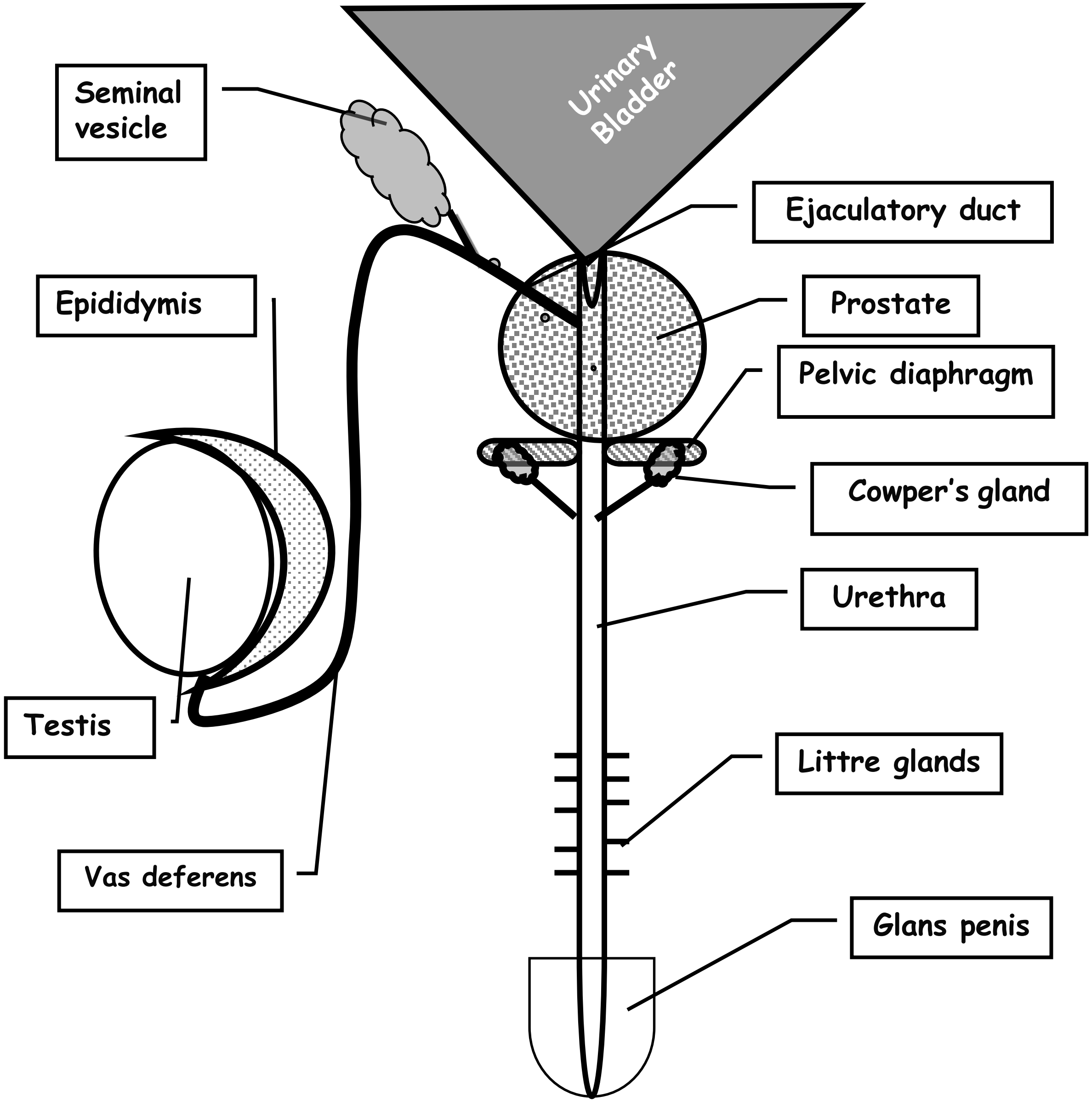
Front View

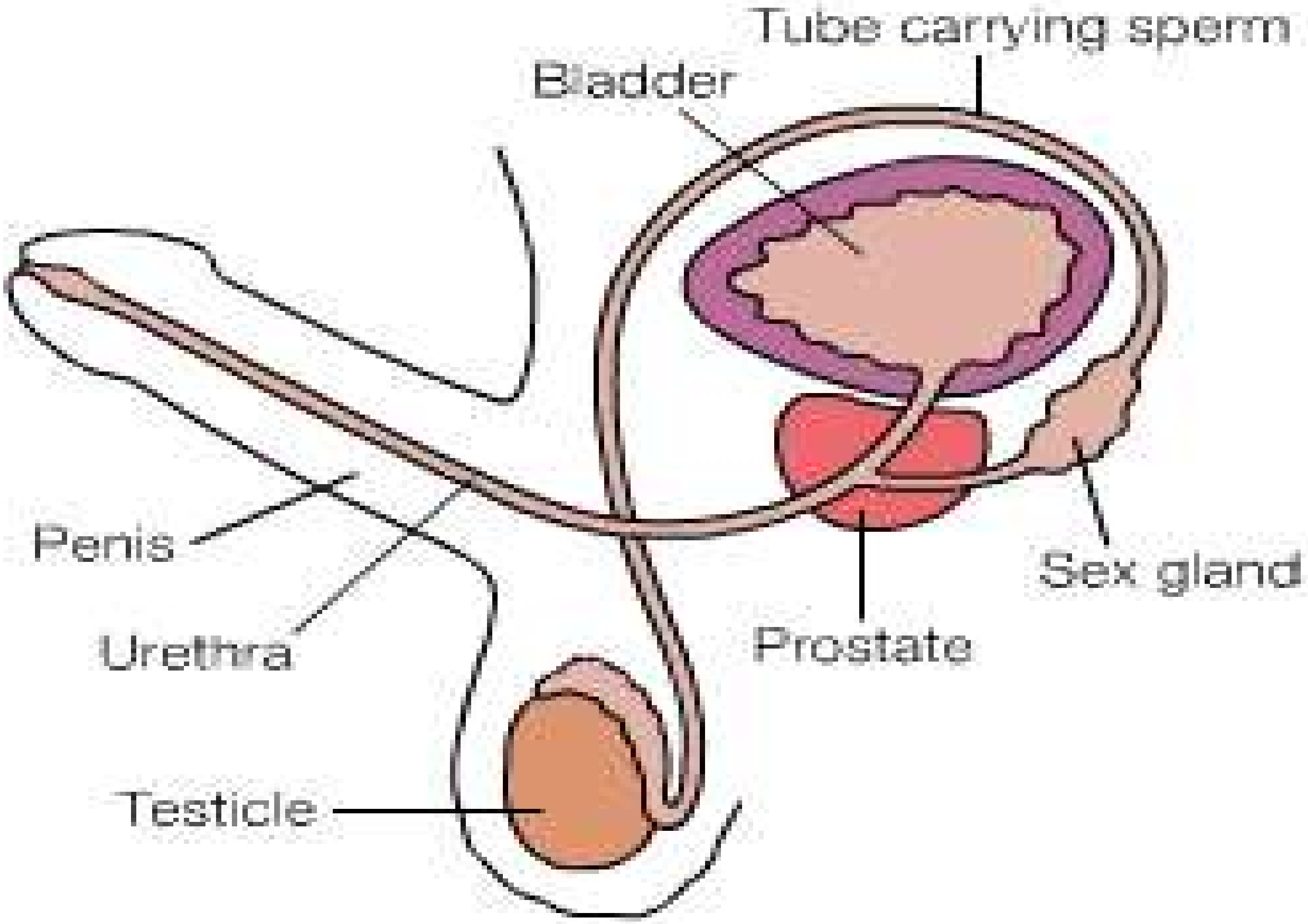
Side View

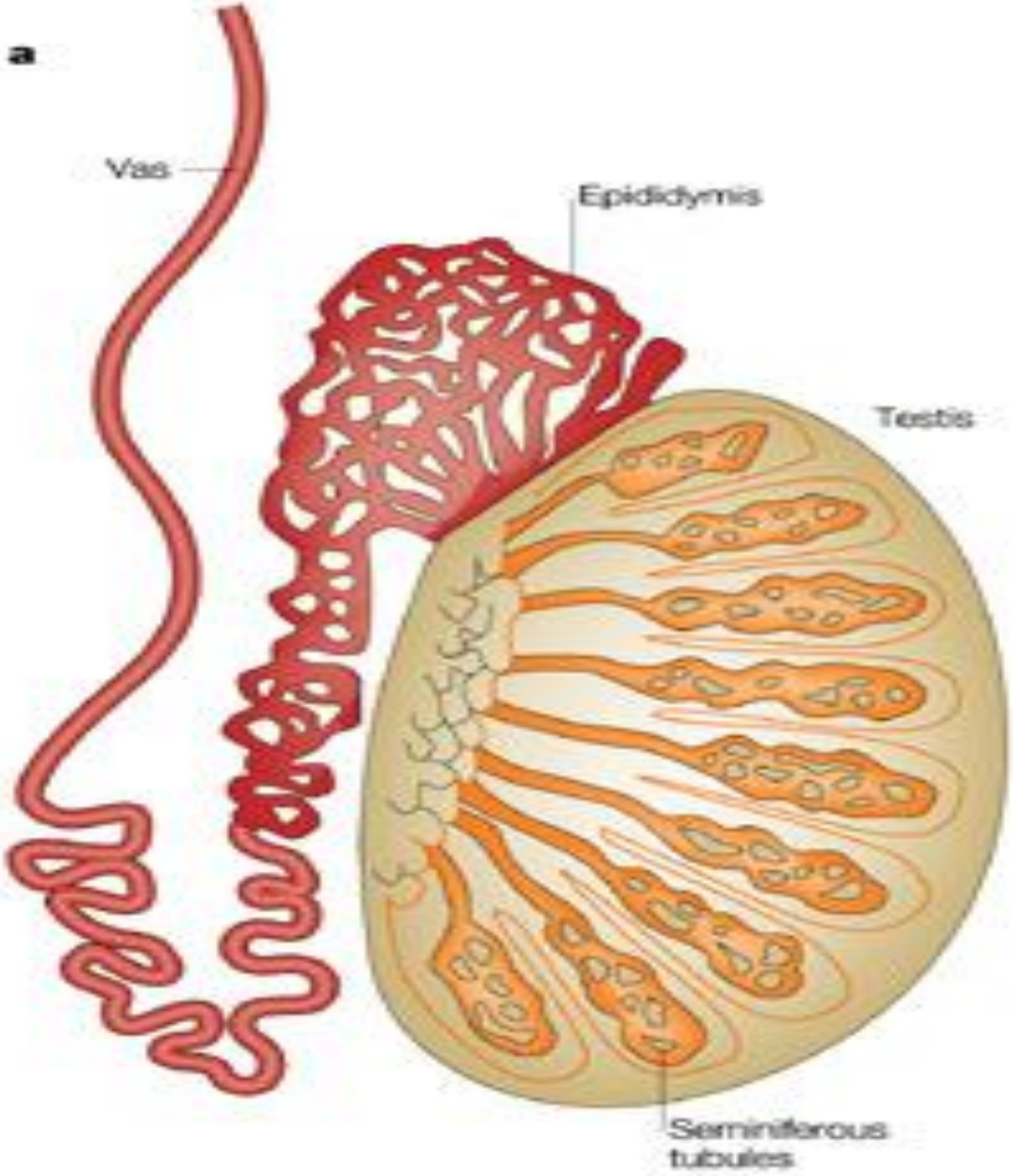
Ampulla of Vas Deferens
Bladder
Prostate Gland
Seminal Vesicle
Ejaculatory Duct
Vein
Artery
Cowper's Gland
Vas Deferens
Skin of Penis
Spermatic Cord
Corpus Cavernosum
Corpus Spongiosum
Testicle, uncovered
Penis
Seminiferous Tubule
Coiled Epididymis
Epididymis
Glans
Vas Deferens
Bladder
Seminal Vesicle
Urethra
Pubic Bone
Prostate
Penis
Epididymis
Testicle
Scrotum
Coronal Ridge
Frenulum

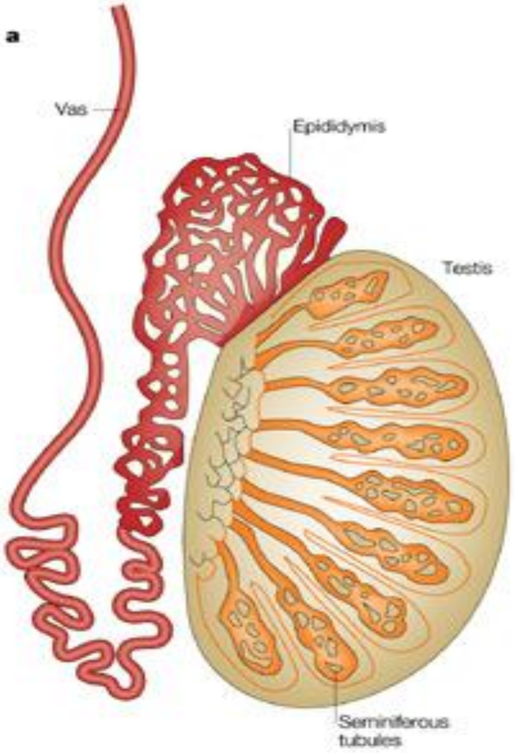
Figure 8-2. Male Anatomy

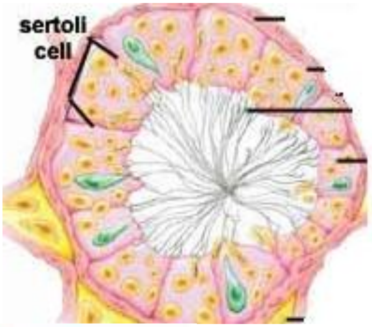

right
ductus
(vas)
deferens
hipbone
seminal vesicle
seminal vesicle duct
prostate gland
ejaculatory duct
urogenital diaphragm
bulbourethral

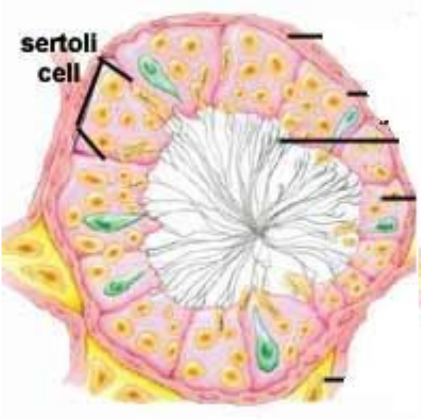

Seminal vesicle
Urinary Bladder
Ejaculatory duct
Epididymis
Prostate
Pelvic diaphragm
Cowper's gland
Urethra
Testis
Littre glands
Vas deferens
Glans penis

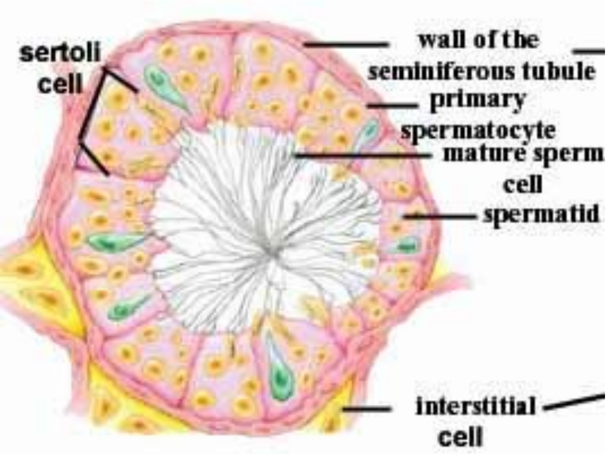

Tube carrying sperm
Bladder
Penis
Urethra
Sex gland
Prostate
Testicle

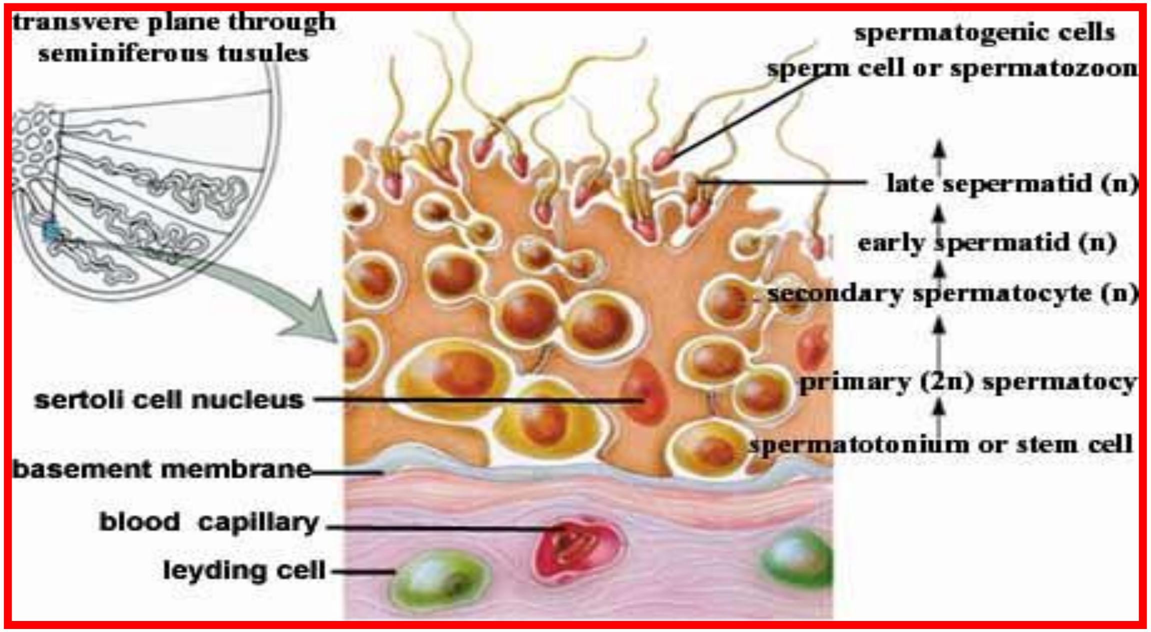

a
Vas
Epididymis
Testis
Seminiferous tubules


a
Vas
Epididymis
Testis
Seminiferous tubules


sertoli cell


sertoli cell

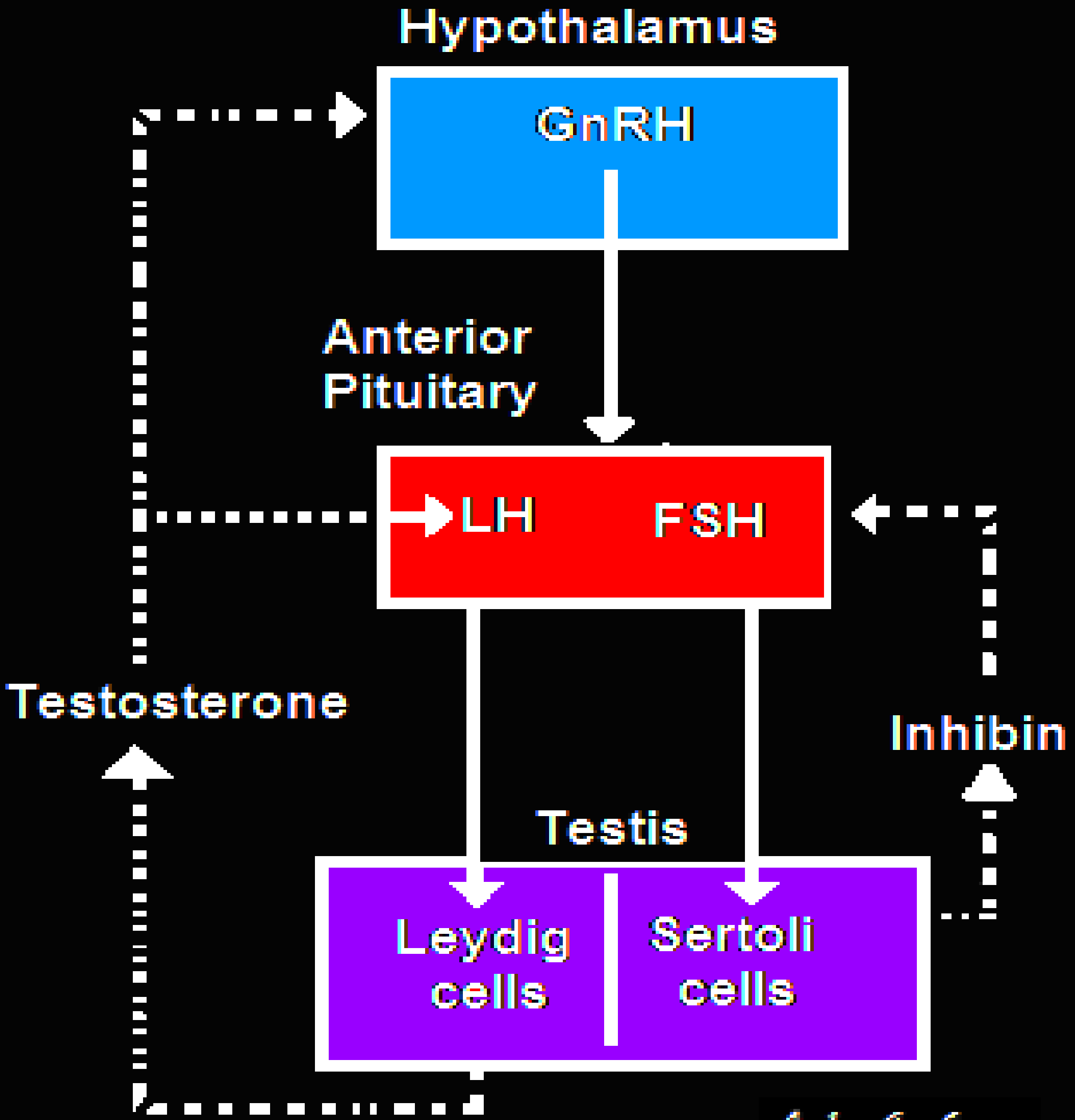

sertoli cell
wall of the seminiferous tubule
primary spermatocyte
mature sperm cell
spermatid
interstitial cell


transvere plane through seminiferous tusules
spermatogenic cells
sperm cell or spermatozoon
late sepermatid (n)
early spermatid (n)
secondary spermatocyte (n)
primary (2n) spermatocy
spermatotonium or stem cell
sertoli cell nucleus
basement membrane
blood capillary
leyding cell

- **The testis has two functional compartments**:
  - The ***seminiferous tubules*** containing the **germ cell** population and the **Sertoli cells**.
  - The ***interstitial compartment*** containing **Leydig cells** responsible for androgen synthesis.

The **hypothalamus** secretes gonadotropin releasing hormone (**GnRH**) stimulating the **pituitary** gland to secrete:

- *Follicle stimulating hormone* (**FSH**): stimulates Sertoli cells to produce androgen binding protein (ABP) and inhibin.
- *Luteinizing hormone* (**LH**): stimulates Leydig cells to produce testosterone.



Hypothalamus

GnRH

Anterior
Pituitary

LH

FSH

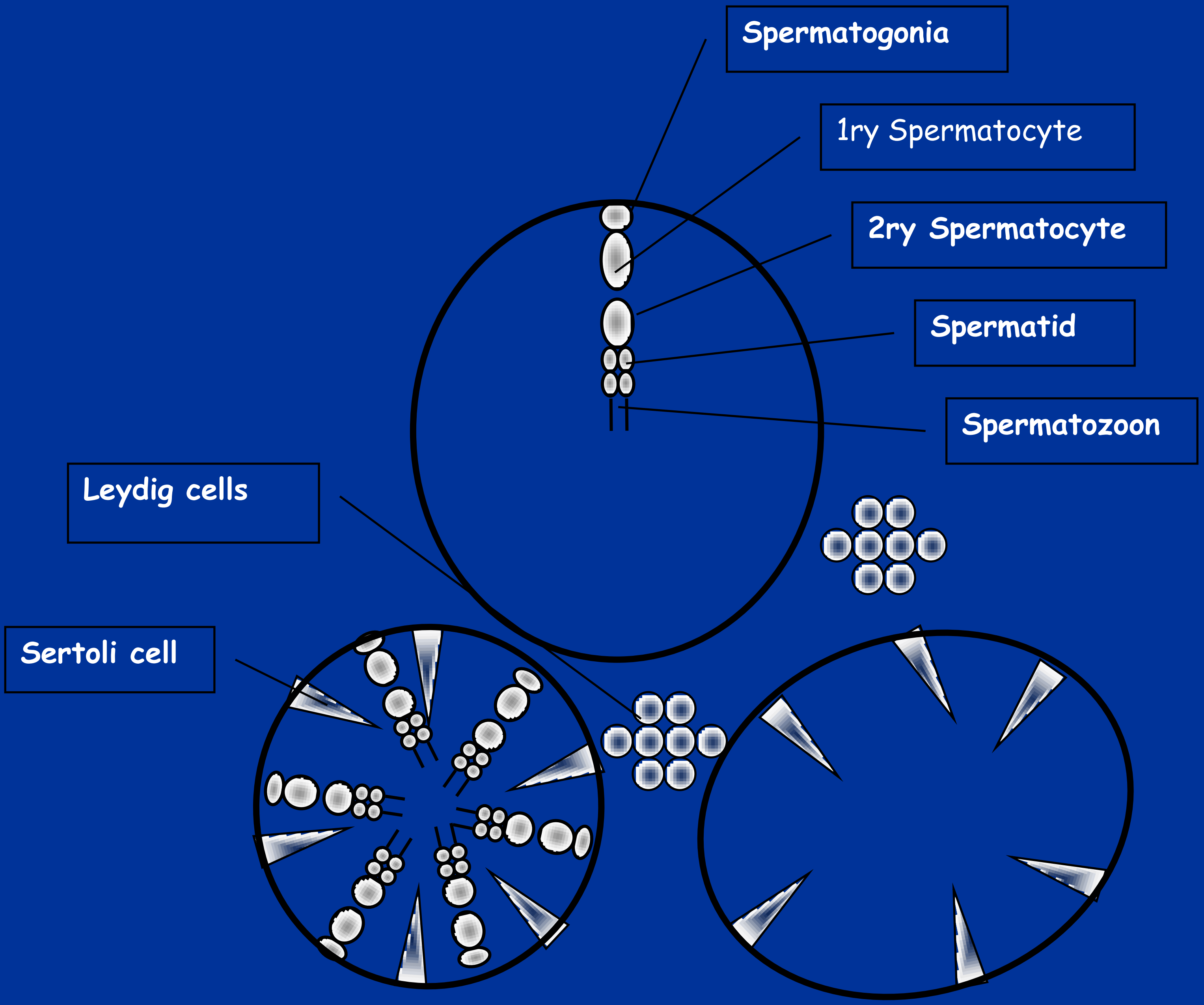
Testosterone

Inhibin

Testis

Leydig
cells

Sertoli
cells



Spermatogonia

1ry Spermatocyte

2ry Spermatocyte

Spermatid

Spermatozoon

Leydig cells

Sertoli cell

ABP carries testosterone to the seminiferous tubules to stimulate **SPERMATOGENESIS**, which proceeds as follows:

Spermatogonia (46 chromosomes) present at the basement membrane of the seminiferous tubule divide mitotically giving rise to ***primary spermatocytes*** (46 chromosomes), which undergo meiosis to be transformed into ***secondary spermatocytes*** (23 chromosomes) then to ***spermatids*** (23 chromosomes).

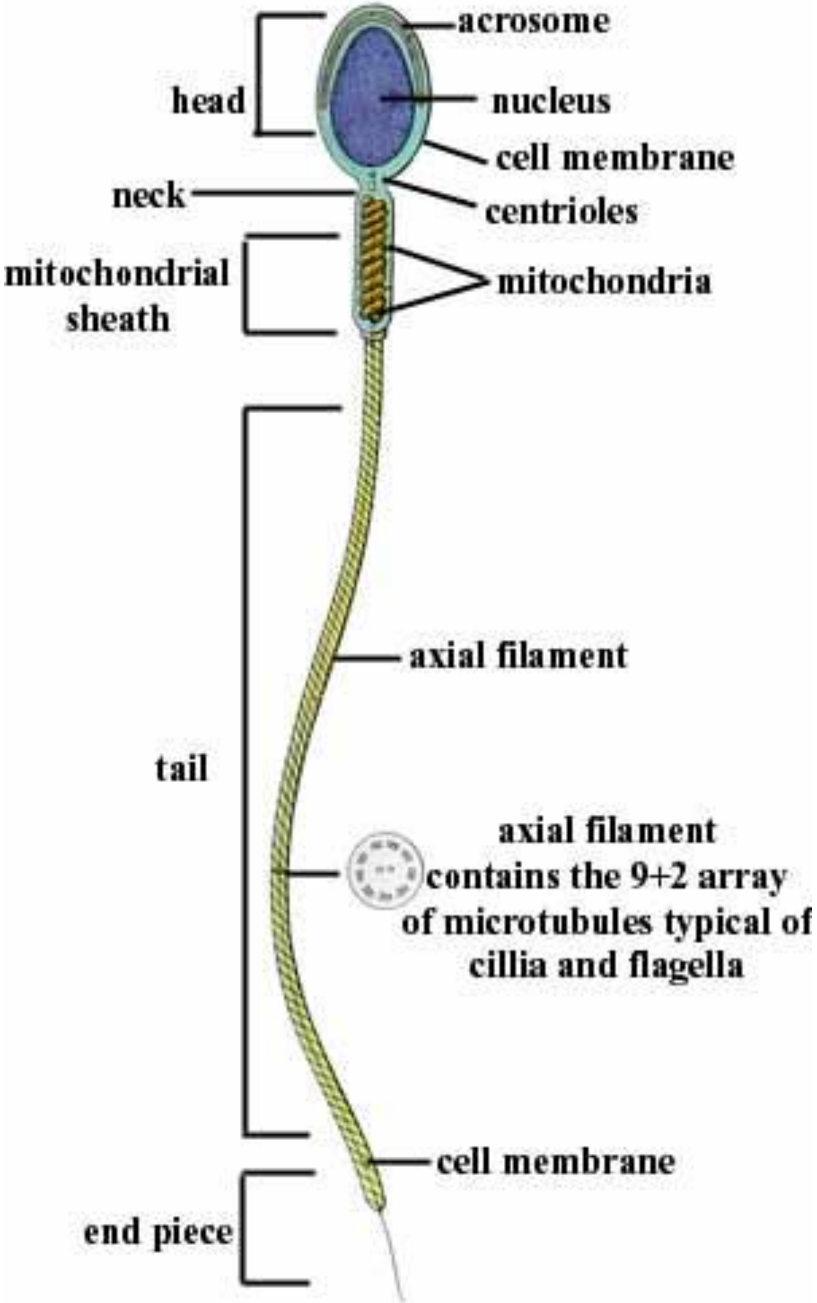
Spermatids are round cells that undergo a process called **SPERMIOGENESIS**, which is a metamorphosis (change of shape) to give rise to the spermatozoon, which have the characteristic shape (head, midpiece and tail).

- From the lumen of the seminiferous tubules, sperms pass to the rete testis then along the vasa efferentia to the epididymis.

- The epididymis is a single highly convoluted tubule, about 5-6 meters long. Sperm transport in the epididymis takes 2-3 weeks, a period essential for sperm maturation.

- At ejaculation, spermatozoa stored in the tail of the epididymis are expelled by the peristaltic waves along the vas deferens to the posterior urethra.

- Seminal plasma volume is mainly formed by the seminal vesicles (60%) and the prostate (30%). The spermatozoa and the epididymal plasma form the remaining 10%.


acrosome
head
nucleus
cell membrane
neck
centrioles
mitochondrial sheath
mitochondria
axial filament
tail
axial filament contains the 9+2 array of microtubules typical of cillia and flagella
cell membrane
end piece

Male infertility

- Definition
- Types
- Causes
- Diagnosis (Evaluation)
- Treatment:
  - Surgical
  - Medical (Current, Future)

Definition of Infertility ?

- **Fertility** = Capability to conceive or induce pregnancy.
- **Infertility**: failure to conceive after **one** year of **unprotected** **regular** sexual intercourse. (While 85% of couples are able to conceive after 1 year of unprotected sexual intercourse, 15% of them are unable to do so).
- **Subfertility** = The probability of spontaneous conception may be decreased but not reduced to zero.
- **Sterility** = Permanent & irreversible infertility.

Types of Infertility

- ***Primary Infertility***: no pregnancy at all has been achieved.

- ***Secondary Infertility***: pregnancy had occurred and no further pregnancy (irrespective of whether she is the present partner, and irrespective of the pregnancy outcome).

Causes of Infertility	%
Male factors alone*	30
Female factors alone**	40
Combined	20
Unexplained (unknown cause)	10-15

*Could be **Pre-testicular**, **Testicular** or **Post-testicular causes**.
Could be **Ovarian, **Tubal**, **Uterine**, **Cervical** or **Peritoneal** factors.

Causes of Male Infertility

Pre-testicular	Hormonal	-Hypothalamic (e.g.Kallman synd) → **Hypo/hypo** -Pituitary (e.g.hyperprolactinemia) -Thyroid -Suprarenal
	Systemic	-Hepatic -Renal, Respiratory, Pancreatic. -Malignant
Testicular	Congenital	-KF synd (47XXY) →**Hyper/hypo** -SCO synd, Maturation arrest -Cryptorchidism -Sperm disorders (Abnormal morphology; e.g.Globozoospermia, or ↓motiliy; e.g.Imm.cilia synd)
	Traumatic	-Excess heat (occupation, sauna) -Testicular op. -Gonadotoxins (drugs, irradiation)
	Inflammatory	-Mumps orchitis
	Neoplastic	-Testicular Ts
	Vascular	-Varicocele -Testicular torsion
Post-testicular	*Mechanical* (P.curvature,PE,RE,Aspermia,ED,hypo-epispadius,Obesity) *Obstructive* (Congenital: CAV or Acquired: iatrogenic (Vasectomy, during hernia repair),or Bilh, TB, leprosy, syphilis), Complete or incomplete *Immunological* (Antisperm-Abs) *MAGI* (Epid, vas, Prostatovesiculitis, urethra?)	

- Unexplained (Idiopathic) Infertility

- Genetic & chromosomal disorders (genetic Infertility) = 30% of sever Oligozoospermia and Azoospermia

How to Diagnose Male Infertility ?

How to Diagnose Male Infertility

- **History taking** (including complaint)
  - Personal
  - Family
  - Present
  - Sexual
  - Past
- **Clinical Examination** (General & Local)
- **Investigation.**

Clinical Examination

- **General Examination**:
  - Male secondary sexual characters (hair distribution, voice, muscle development, fat distribution, height : span ratio)
  - Gynecomastia

- **Local Examination**:
  - **Penis:** urethral orifice (hypospadias & epispadias may cause mechanical infertility)

  - **Testis:** if palpable or not, its size & consistency

  (normal consistency= rubbery, normal size: length >3.5cm, width >2.5cm, volume 16-20ml)

  - **Epididymis:** epididymal nodules denote obstruction.

  - **Vas deferens:** if present or absent & and if it is beaded (T.B), thich (β)

  - **Spermatic cord:** for the presence of varicocele

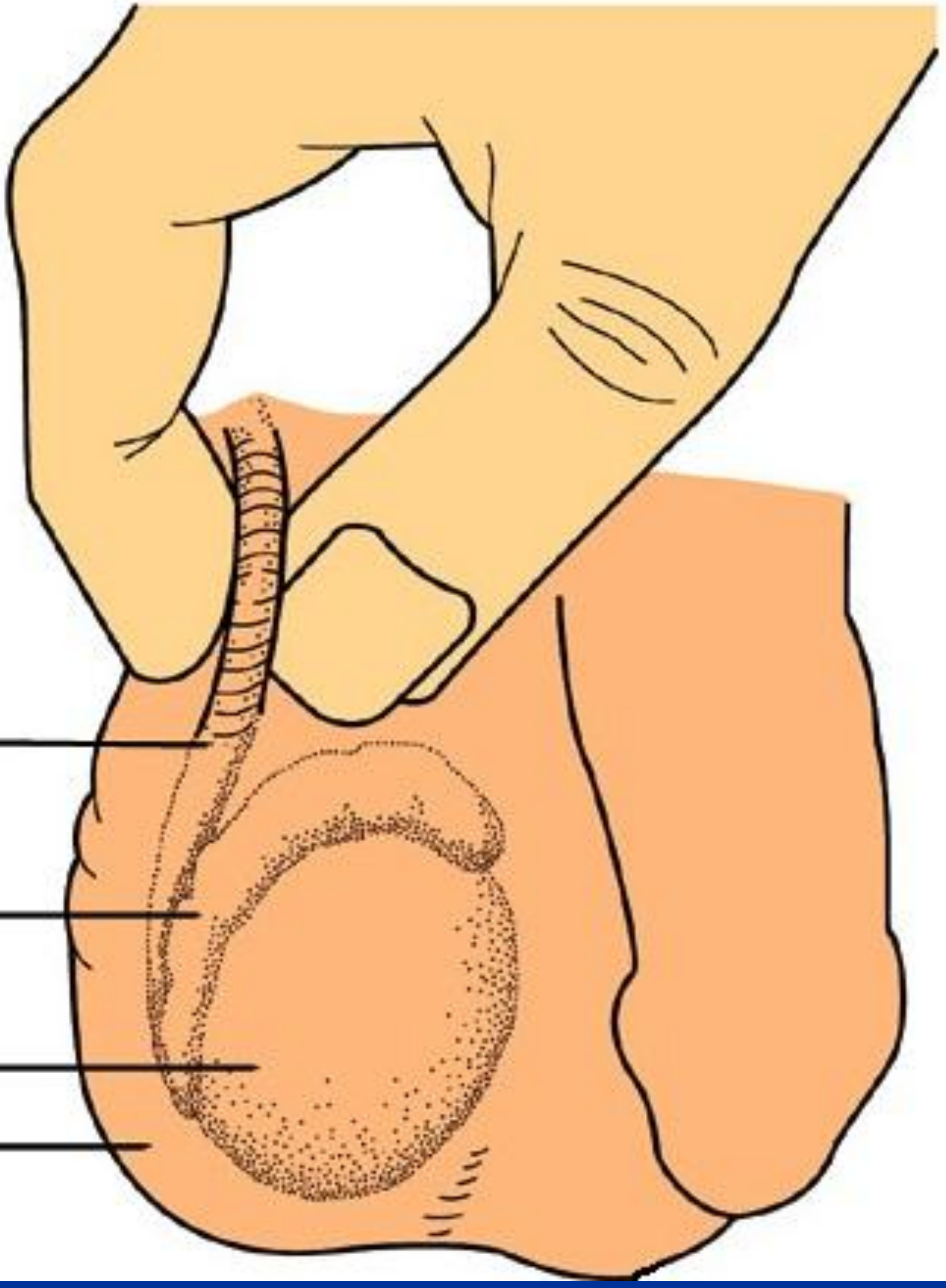
  - **PR examination:** for prostate (size, consistency & surface regularity) & seminal vesicles (normally not felt)

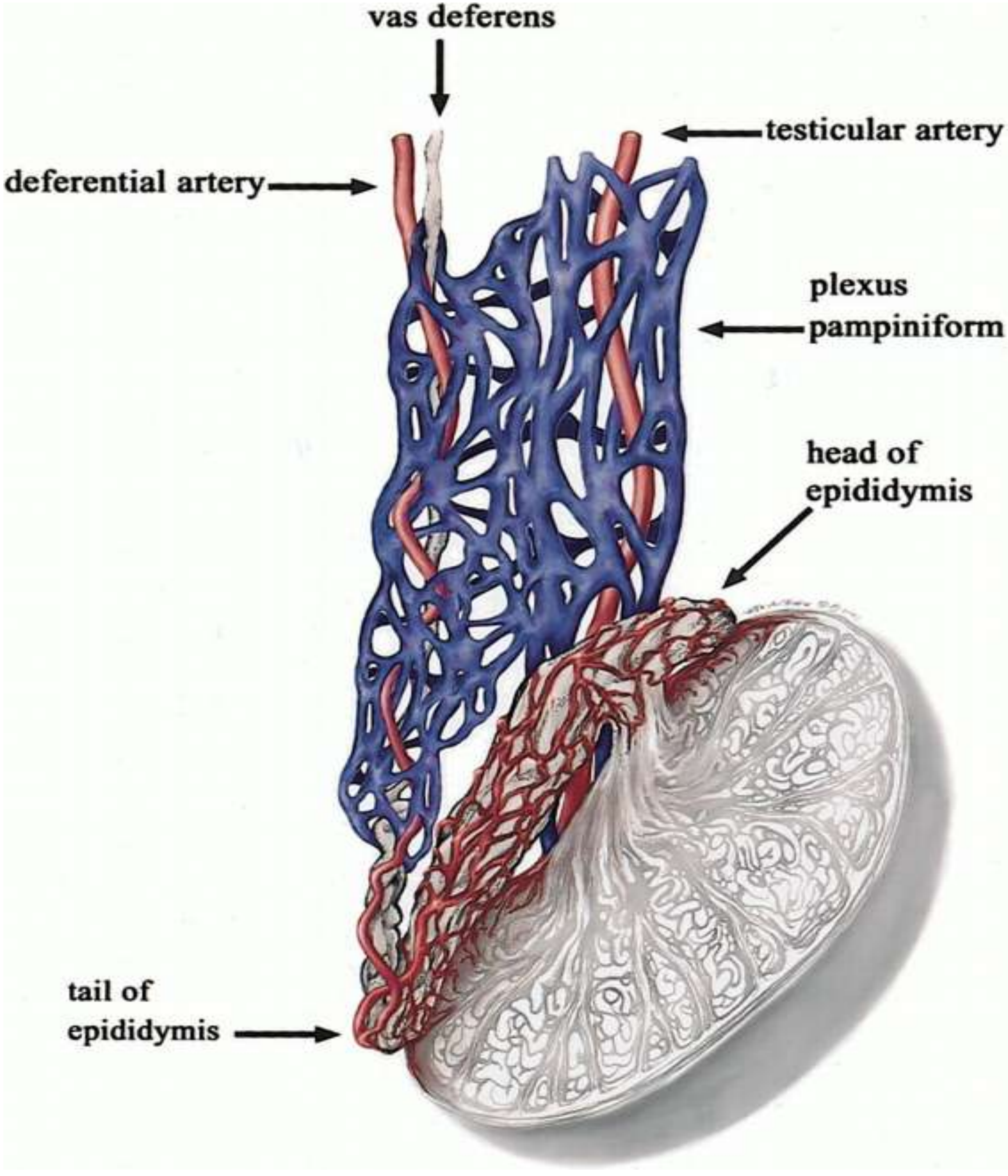
General Examination

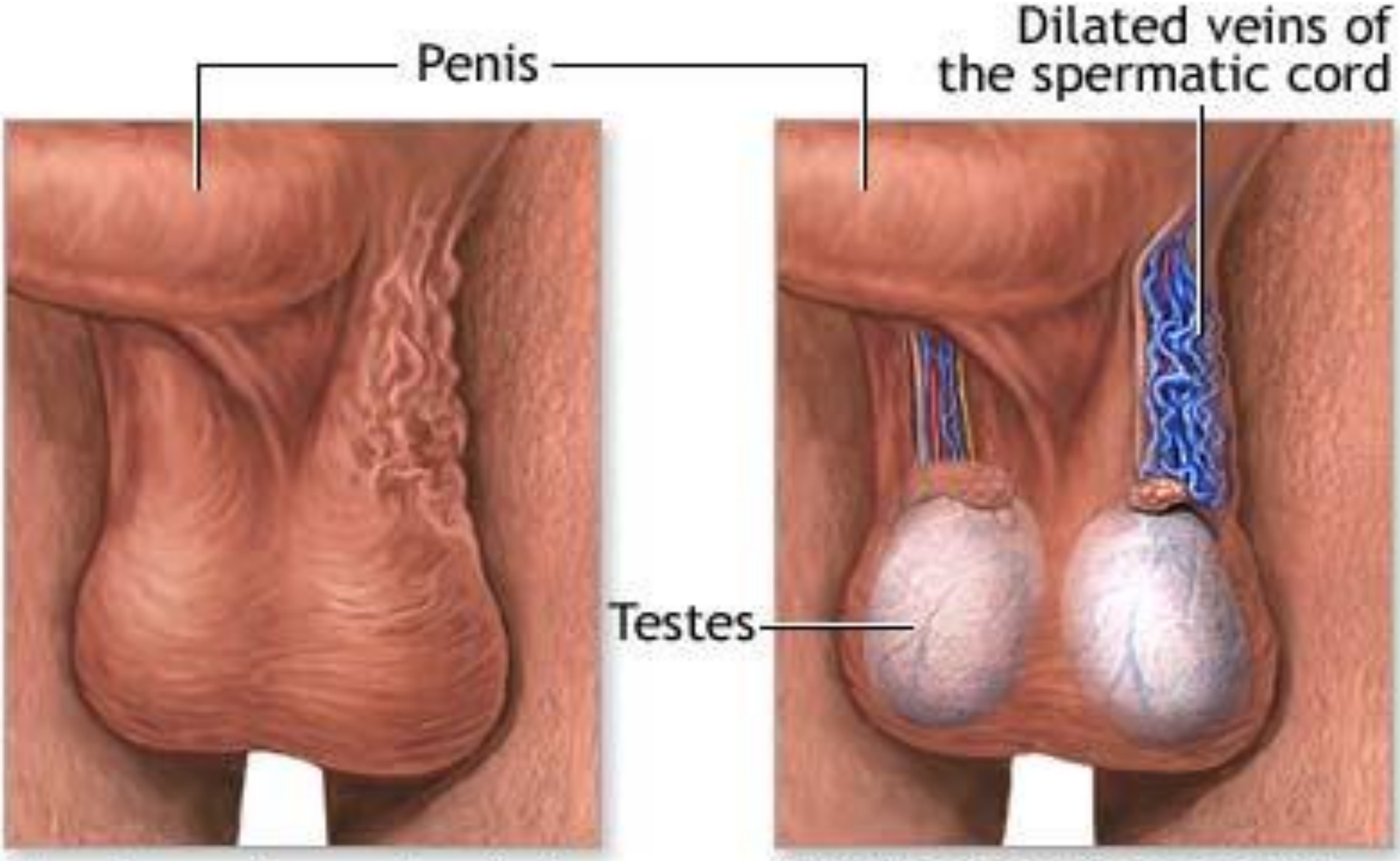
Manifistations of male hypogonadism:

1. patient usually is tall
2. sparse hairs
3. flat topped pubic hair , no frontoparietal recession
4. gynaecomastia
5. limbs maybe disproportion (Span >height, LS >US by 5cm).
6. flabby muscles

The Scrotal Structures


Spermatic cord
Epididymis
Testis
Scrotum


vas deferens
testicular artery
deferential artery
plexus pampiniform
head of epididymis
tail of epididymis


Penis
Dilated veins of the spermatic cord
Testes

A varicocele can be felt and sometimes be seen as a tortuous mass on the surface of the scrotum

A varicocele is made up of veins that contain inadequate valves

A.D.A.M.

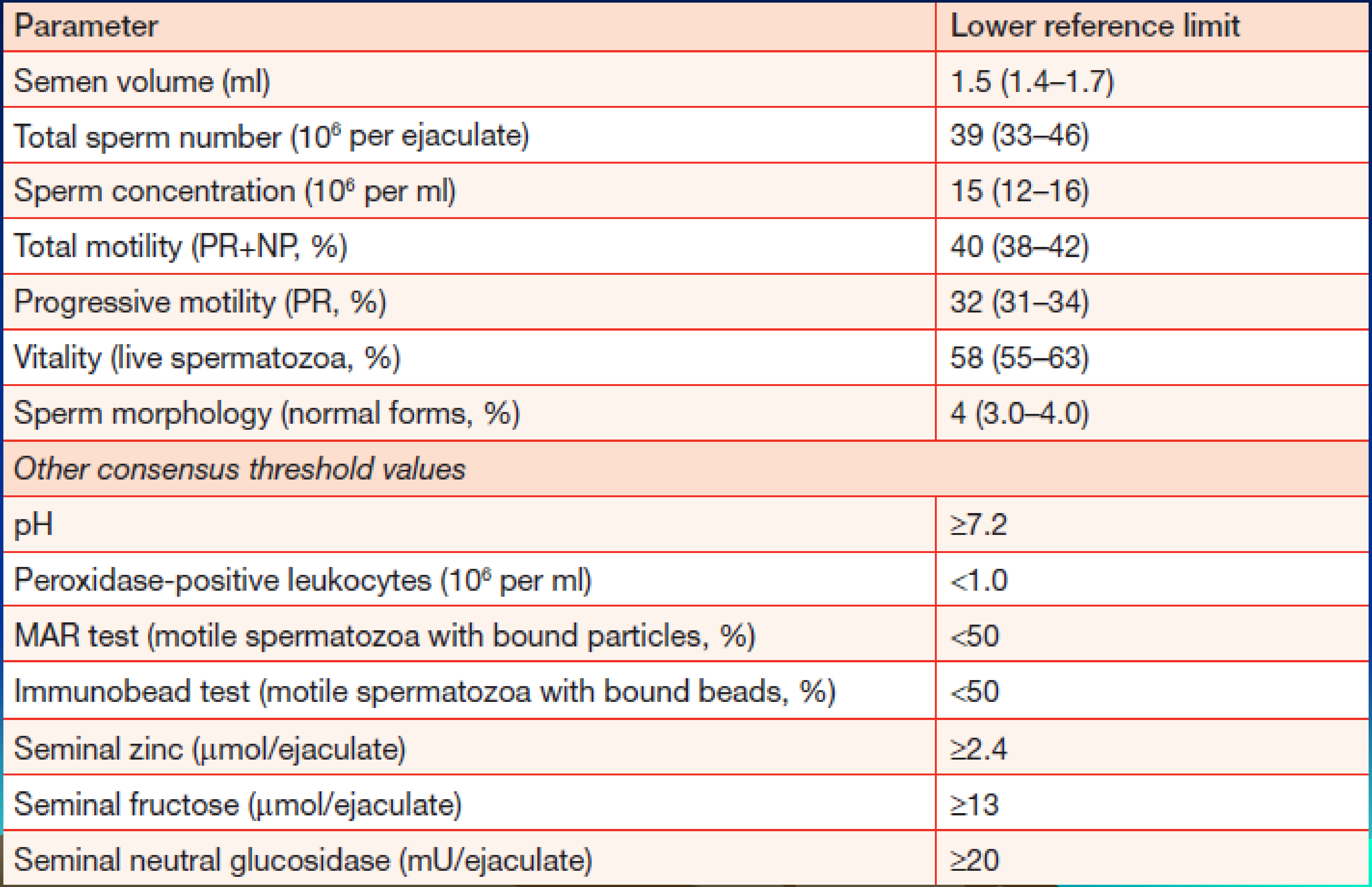
Investigation

- Semen analysis
- Urine & prostatic discharge analysis
- Hormonal Profile (FSH, LH, T, PRL, others e.g.TSH)
- Imaging (Scrotal US & Duplex, TRUS)
- Testicular biopsy (???)
- Other specific investigations (e.g., Antisperm antibodies, Electron microscopy, Sperm function tests, ….)
- Chromosomal study (Karyotyping), Genetic study.

Semen Analysis

- **Color**: grayish-white
- **Volume**: 2ml or more (**60% seminal vesicles, 30% prostate & 10% epididymis & testis**).
- **PH**: 7.2 or more (alkaline)
- **Liquefaction time**: less than 30 minutes, **Viscosity**: Normal, how?
- **Sperm concentration/ml**: more than 20×10^6/ml (& less than 250 millions/ml)
- **Sperm motility**: 50% or more of the spermatozoa show progressive motility (grades a & b) or 25% or more with rapid linear progression (grade a) wihin 60 minutes of ejaculation
- **Sperm morphology**: Normal forms at least 30% (WHO), or 14% (Kruger strict criteria).
- **Vitality**: 75% or more live. i.e. excluding dye.
- **WBCs**: fewer than 1×10^6/ml
- **Antisperm Antibodies**: fewer than 50% motile spermatozoa with beads (Immunobead test) or adherent particles (MAR test)
- **Biochemical markers (not routine):**
  - Fructose estimation (seminal vesicle marker): if bilateral congenital absence of the vas deferens is suspected.
  - L-Carnitine estimation (epididymal marker): may help differentiating pre from post-epididymal obstruction.

Semen analysis (WHO, 2010)



Parameter	Lower reference limit
Semen volume (ml)	1.5 (1.4–1.7)
Total sperm number (10^6 per ejaculate)	39 (33–46)
Sperm concentration (10^6 per ml)	15 (12–16)
Total motility (PR+NP, %)	40 (38–42)
Progressive motility (PR, %)	32 (31–34)
Vitality (live spermatozoa, %)	58 (55–63)
Sperm morphology (normal forms, %)	4 (3.0–4.0)
Other consensus threshold values	
pH	≥7.2
Peroxidase-positive leukocytes (10^6 per ml)	<1.0
MAR test (motile spermatozoa with bound particles, %)	<50
Immunobead test (motile spermatozoa with bound beads, %)	<50
Seminal zinc (µmol/ejaculate)	≥2.4
Seminal fructose (µmol/ejaculate)	≥13
Seminal neutral glucosidase (mU/ejaculate)	≥20

- How to collect the sample ?????
- If **Normal** semen analysis → no need to repeat it.
- If **abnormal** or Azoospermia → Repeat

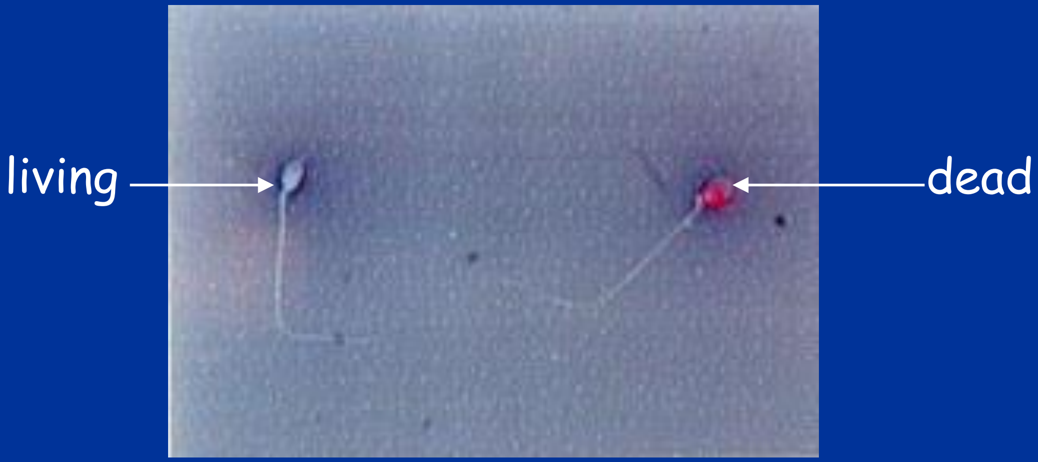
Low semen volume or Normal volume ????

Semen Volume: (60% seminal vesicles, 30% prostate & 10% epididymis & testis):

- Short abstinent period (N= 2 - 7 days).
- Lost portion.
- Coitus interruptus.
- Idiopathic.
- Pathological causes (Hypogonadism, EDO, CAV, Retrograde ejac., Infection & fibrosis).
- Khat (Qat) chewing????

Immotile or Dead sperms (Necrozoospermia) ?????

- Diff. by Eosine stain, or HOST.


living
dead

WBCs or Spermatogenic cells (Round Cells) ?????

- Diff. by:
  - Pyroxidase test: depends on the presence of peroxidase enzyme in PMNL, but cannot detect activated leukocytes or lymphocytes.
  - Monoclonal antibodies (antigen CD45).

Nomenclature

- Low semen volume (oligospermia)
- Aspermia
- Azoospermia (???? Cryptozoospermia): Obstructive or Functional)
- Oligozoospermia
- Asthenozoospermia
- Teratozoospermia
- OAT
- Necrozoospermia.
- Leucocytospermia (STDs & Non-STDs)
- Hematospermia

Congenital Bilateral Absent Vas (CBAV)

-A clinical diagnosis + Normal sized testes

-Normal Hormones

-Semen picture:

a. **low** semen volume
b. **Acidic** PH
c. absence of prostaglandin and **fructose** in semen. [S.V. Markers]
d. absence of coagulation of semen.
e. absence of sperms [**Azoospermia**]

-IVP or Renal US, TRUS

Haemospermia:

Definition: presence of blood in semen.

Etiology

- **Idiopathic** : the most common.
- **Systemic**: Bleeding disorders, Vit. Deficiency, Drugs, HTN.
- **Local**:
  - Infection (30%) : prostato-vesiculitis, Bilharziasis.
  - Prostatic calculi (20%).
  - Malignancy (15%).
  - Angiomatous malformation.

Hormonal Profile

(FSH, LH, T, PRL, E_2)

(others accordingly: e.g.TSH)

- High FSH ,LH + Small testes = **Hypergonadotrophic Hypogonadism** (primary testicular cause).

- Low FSH, LH and T + Small testes = **Hypogonadotrophic Hypogonadism**

Biochemical markers

- Seminal vesicle markers: **Fructose**, PGs
- Prostatic markers: Zinc, citric acid, acid phosphatase.
- Epididymal Markers: Alpha glucosidase, L-carnitine, glycerophosphocholine.
- Testicular markers: ??????

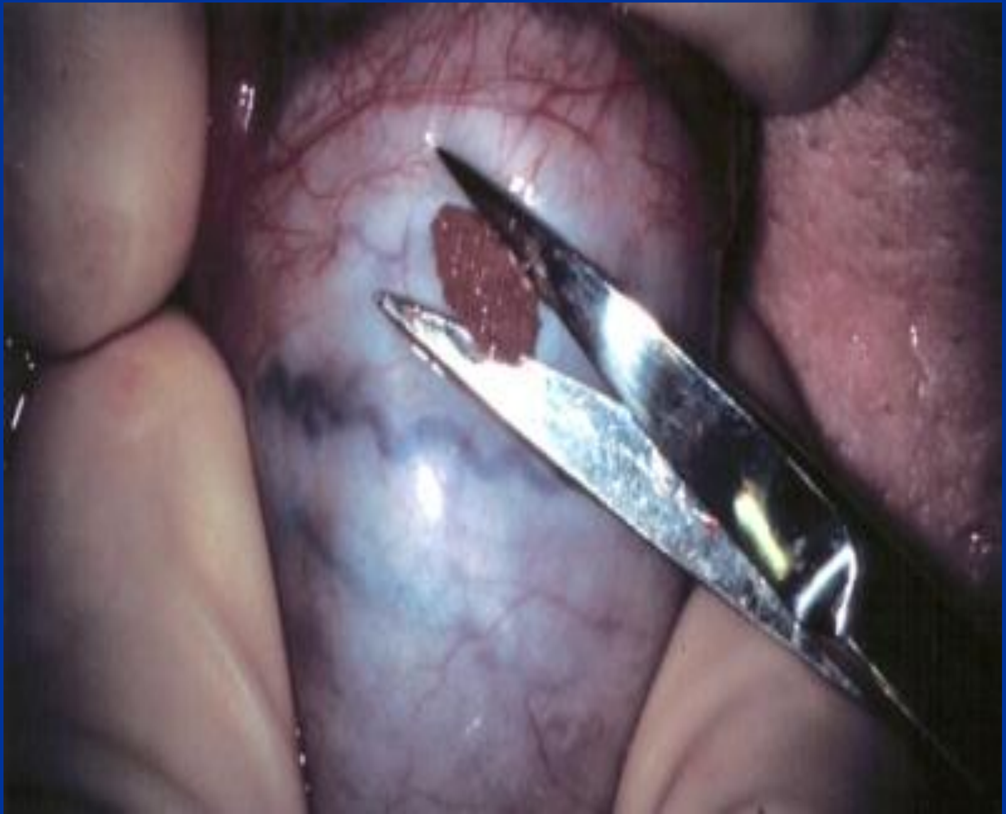
Imaging

- Scrotal US and Duplex
- TRUS
- Others (Seminal vesiculography, Vasography???, MRI, CT scan…..)

Testicular biopsy

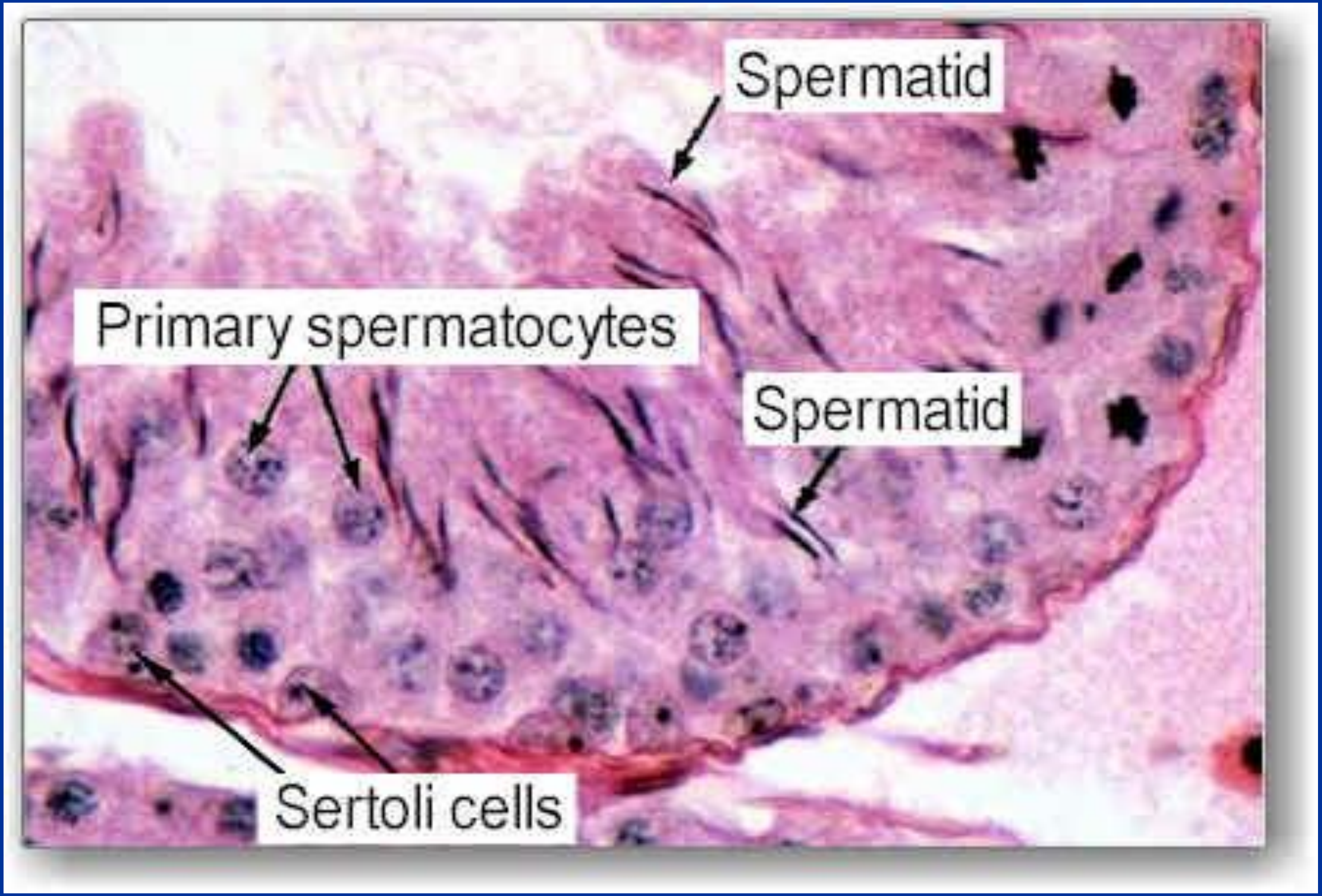
-Diagnostic (Diff. Obstructive From Functional Azo.,

-Therapeutic: (TESE etc…)

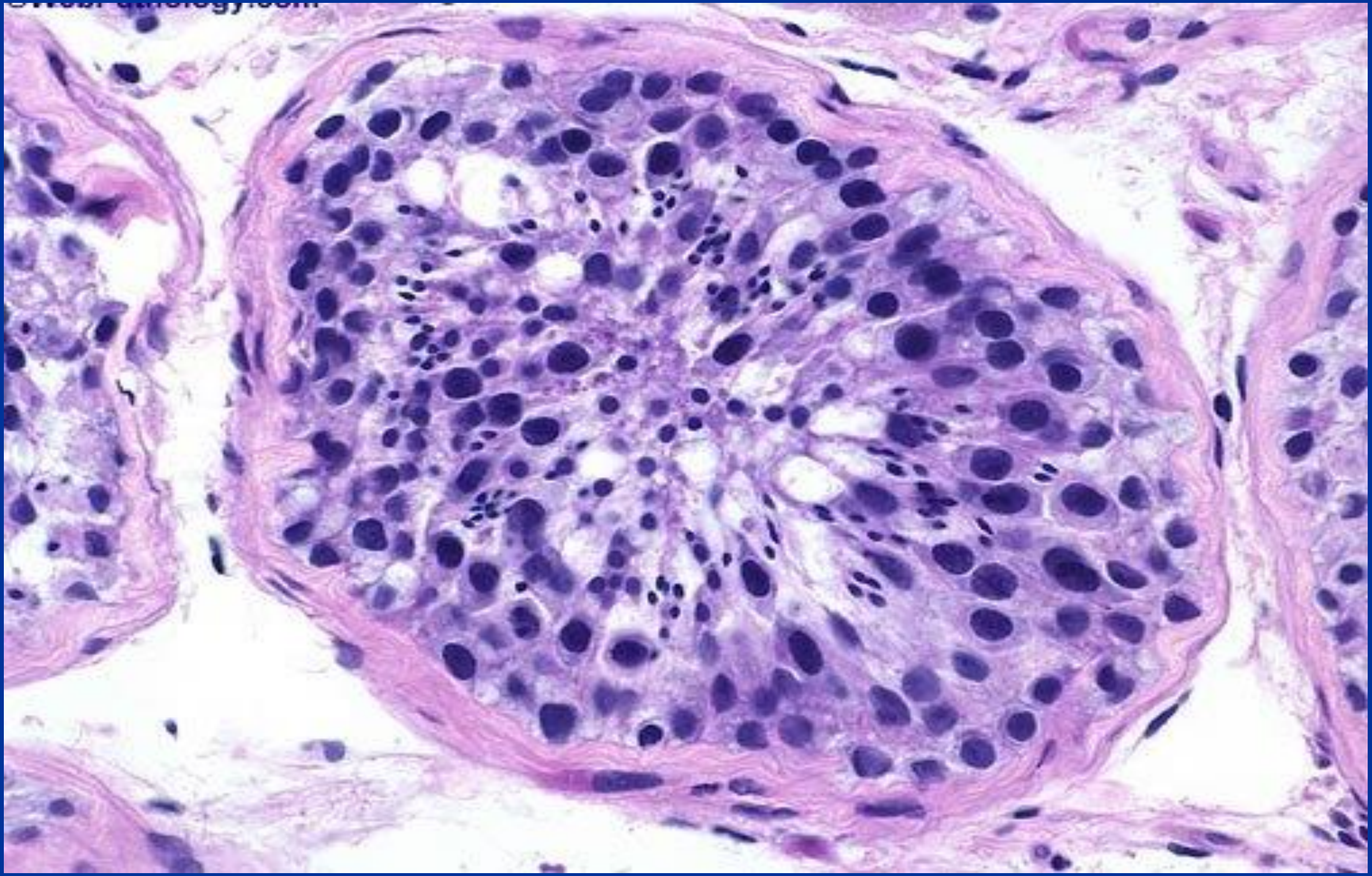


Testicular biopsy

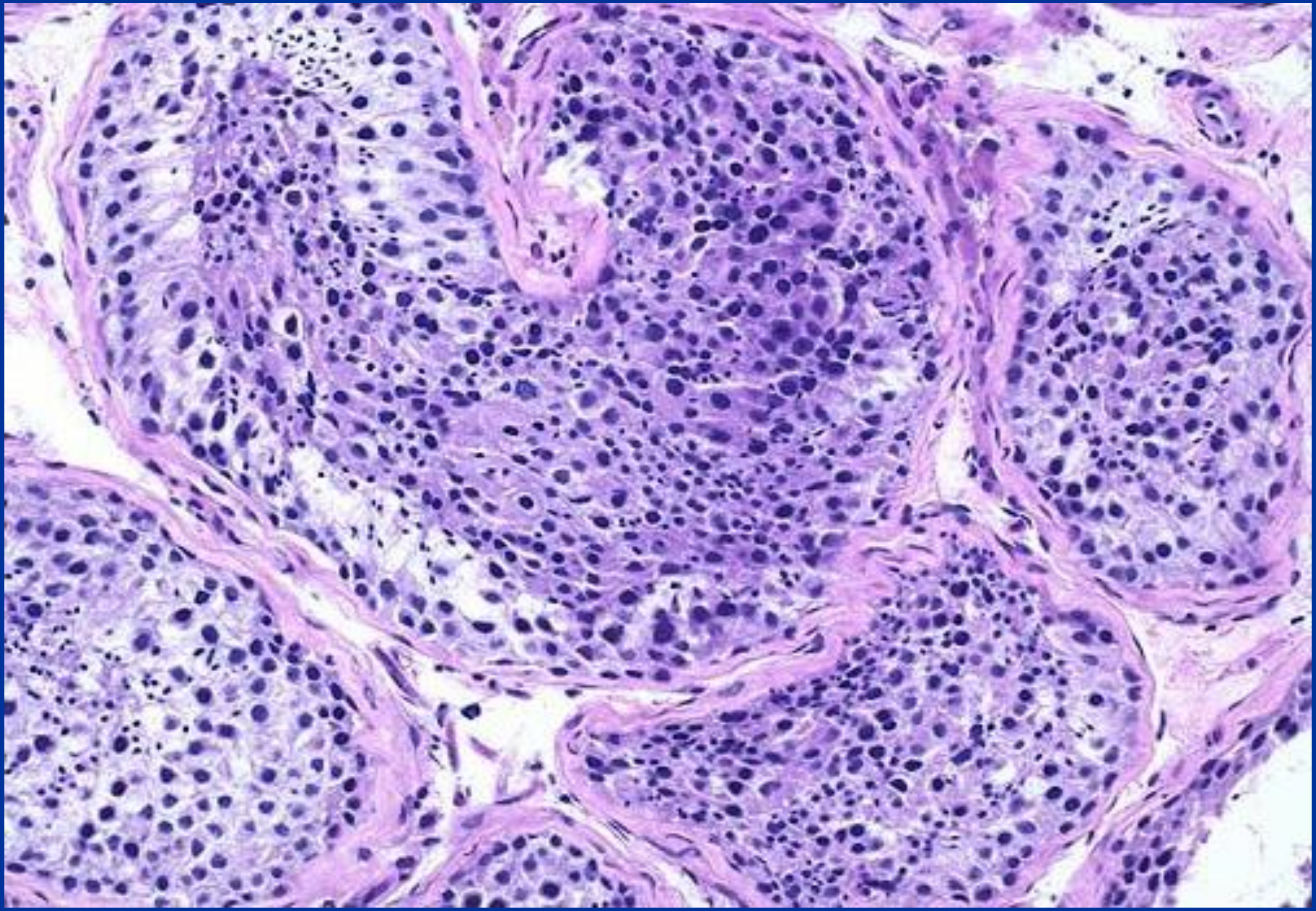
- Opened →TESE (Conventional or Microsurgical)
- Closed (TESA, FNA)
- If sperms present → Cryopreserved for ICSI + H/P.


Spermatid
Primary spermatocytes
Spermatid
Sertoli cells

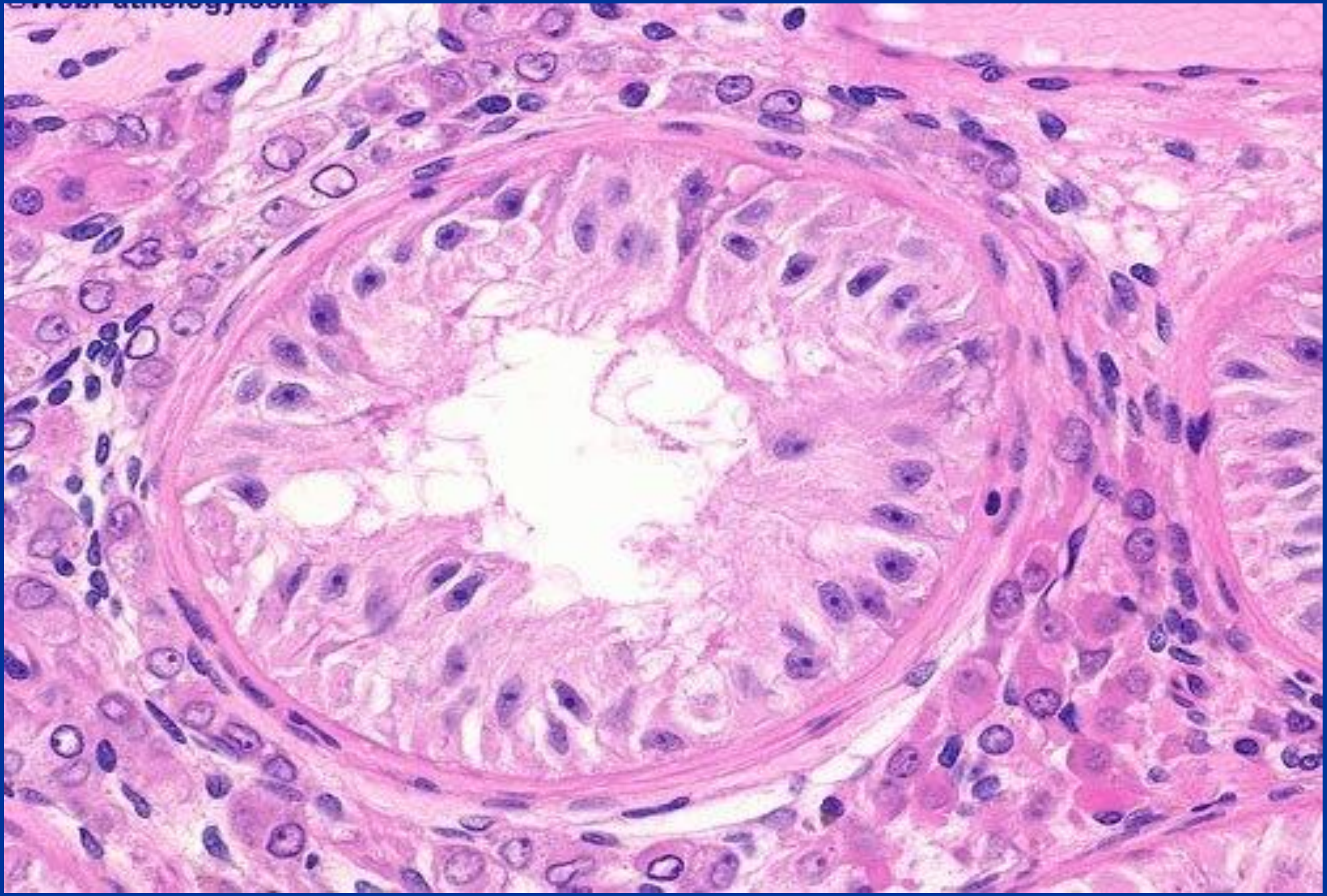
Normal spermatogenesis



Normal spermatogenesis



SCO syndrome



- **Histopathological types**

  -Complete spermatogenesis (Normal)

  -Hypospermatogenesis (<5-10)

  -Maturation arrest (spermatid, spermatocytes, spermatogonia)

  -Sertoli cell only (Germ Cell Aplasia)

  -Complete tubular hyalinization (No cells)

  -Combined.

Genetic analysis & Chromosomal study

- Example: Kleinefelter syndrome with 47XXY (mainly)

Treatment of Male Infertility

- **Prevention** (Surgical, Medical)
- **Medical** (Etiological, Empirical)
- **Surgical** (general, Local)
- **Assisted Reproductive Technology (ART)**
  - Artificial Insemination (e.g., IUI).
  - In Vitro Fertilization (IVF).
  - Gamete micromanipulation (ICSI)
- **Future Therapies** (Cloning, Stem cells, Germ cell culture, Gene therapy)
- **Counseling** (in case of genetic or chromosomal errors)…….**PGD**???

Prevention

- Early surgery for undescended testis (before the age of 1-2 years).
- Early correction of testicular torsion (before 4 hours).
- Early management of urogenital infections.
- Avoidance of gonadotoxins e.g. irradiation & gonadotoxic drugs.
- Cryopreservation of semen before chemotherapy & radiotherapy.

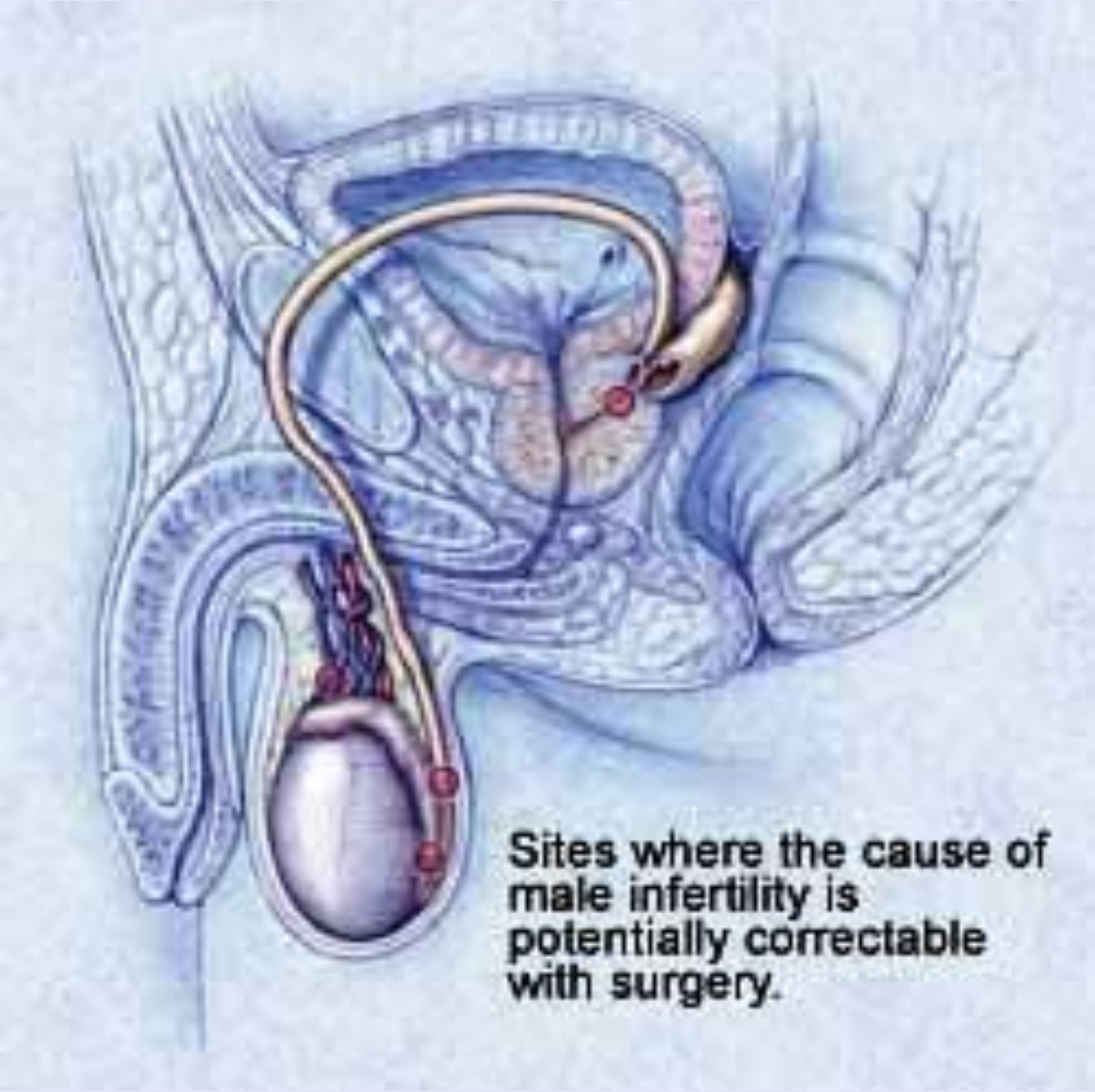
Medical Treatment

Specific Treatment **(for specific disorders) e.g.** **Etiological**	**Non-specific Treatment** **(for idiopathic causes) e.g.** **Empirical**
•Gonadotropins for hypogonadism •Bromocriptine for hyperprolactinemia •Corticosteroids for immune infertility •Antibiotics for infection. •Treatment of systemic diseases e.g., hypo-hyperthyroidism. •Treatment of sexual dysfunction	•Androgen therapy •Anti-estrogen therapy •Human chorionic gonadotropin (HCG) •Human menopausal gonadotropin (HMG). •Antioxidants •Pentoxifylline •L-Carnitine •GH •Others (B12, Zinc, etc) •Herbal

Surgical Treatment

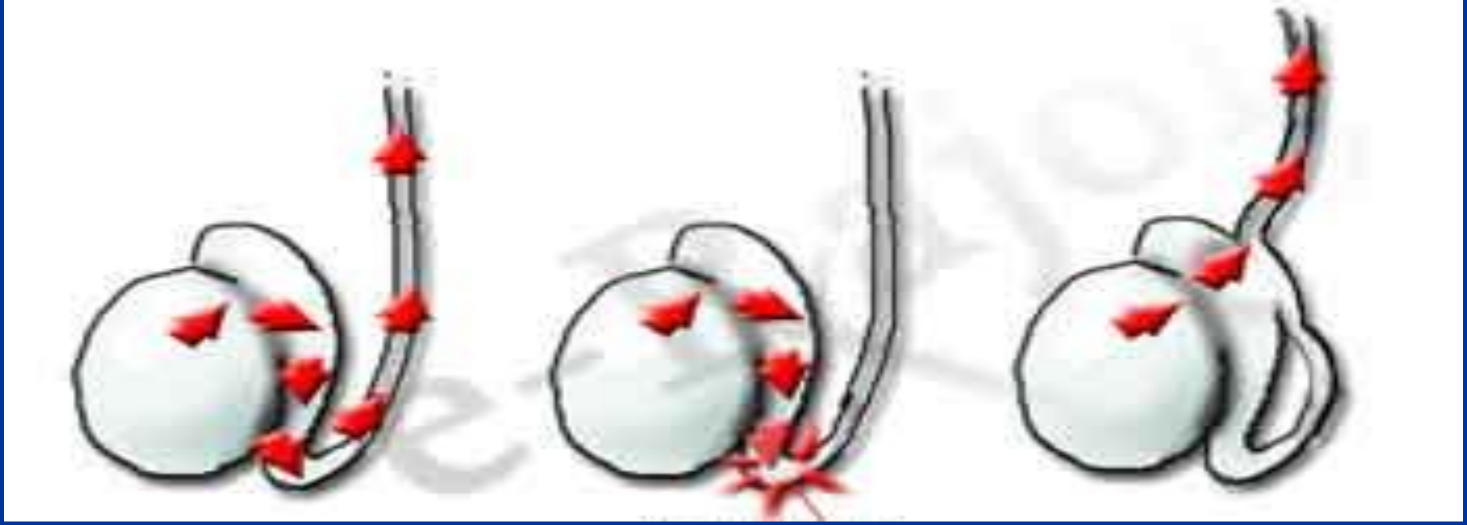
- Varicocelectomy
- Epididymovasostomy, Vasovasostomy.
- TUR-Veru, TUR-ED.
- Vasectomy reversal
- Orchipexy (UT, T Torsion)
- Correction of severe Hypospadius (e.g., perineal)
- TESE, TESA, FNA, PESA

NB: Surgical correction of causes of male infertility may lead to a more aggressive type of infertility.



Sites where the cause of male infertility is potentially correctable with surgery.

Epididymovasostomy



Assisted Reproductive Technology

- **Intrauterine insemination (IUI):** processed semen (including good number and quality of sperms) are deposited in the uterus at the time of spontaneous or assisted ovulation.
- **In vitro fertilization (IVF):** Fertilization is aided by mixing retrieved oocytes with sperms outside the body and incubated to achieve in vitro fertilization.
- **Intracytoplasmic sperm injection (ICSI):** A single normal sperm is injected directly into the oocyte cytoplasm to achieve fertilization.

Indications for Intrauterine Insemination (IUI)

Male factors:

- Mild oligozoospermia, asthenozoospermia, or teratozoospermia.
- Persistent low or high semen volume.
- High semen viscosity & non-liquefaction of semen..
- Retrograde ejaculation (partial or complete)
- Severe premature ejaculation
- Presence of antisperm antibodies.

Female factors:

- Abnormal cervical mucous (chronic cervicitis).
- Immunological reaction of the cervix (hostile cervix with cervical antibodies).

Failure to conceive after ovulation induction.

Unexplained infertility.

Indications for ICSI

Male factors:

- Severe & persistent oligozoospermia and/or asthenozoospermia, and/or teratozoospermia not responding to other measures.
- Azoospermia (obstructive or functional)
- Aspermia.
- Low semen volume (after repeated failure of artificial insemination).
- High semen viscosity & non-liquefaction of semen (after repeated failure of artificial insemination).
- Antisperm antibodies (after repeated failure of artificial insemination).
- Failed surgery to relief obstruction of the genital tract (e.g: epididymovasostomy, vasovasostomy, TUR).

Female factors:

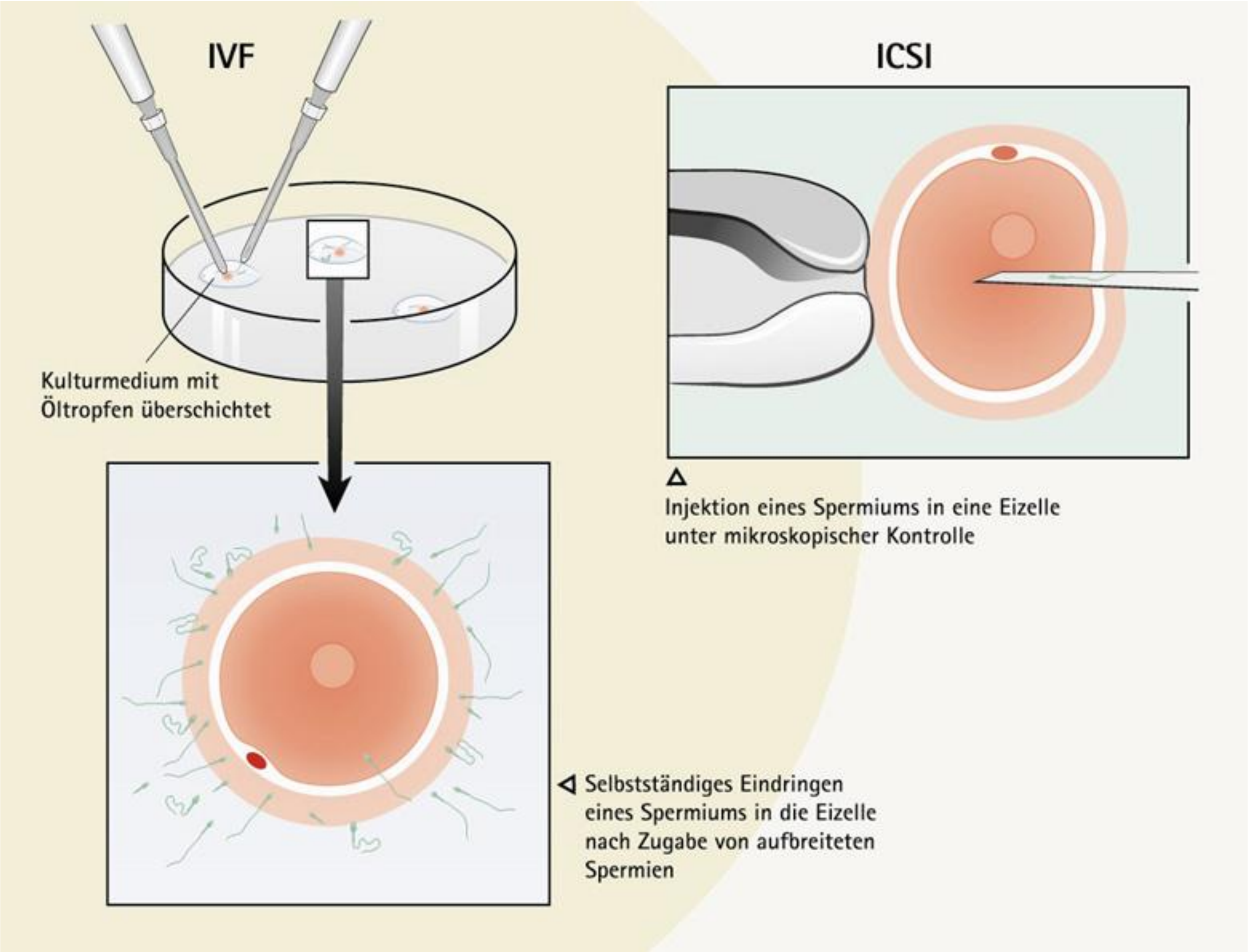
- Tubal obstruction (Bilateral or unilateral).
- Abnormal cervical mucous (chronic cervicitis).
- Immunological reaction of the cervix (hostile cervix with cervical antibodies).

Unexplained (idiopathic) Infertility.

Preimplantation Genetic Diagnosis (PGD).

The Technique of IVF and ICSI

- The ovaries are stimulated by daily injections of gonadotropins to optimize follicular development, which is monitored by means of transvaginal ultrasonography (**Controlled ovarian stimulation**).
- After controlled ovarian stimulation, oocytes are retrieved by means of ultrasound-guided transvaginal aspiration of follicular fluid.
- Recovered oocytes are often fertilized in vitro by culturing them with many motile sperm (**Conventional IVF**)
- Oocytes can also be fertilized by means of intracytoplasmic sperm injection (**ICSI**), a technique in which a single normal sperm is injected into the oocyte.
- Multiple embryos are cultured, often for 3 days or 5 days
- Selected embryos are transferred back to the uterus (**Embryo transfer**).
- Good-quality, excess embryos are often **cryopreserved** to be used for another trial.

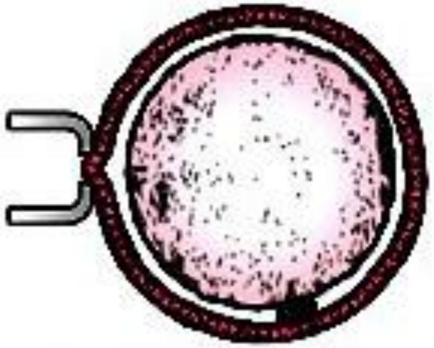

IVF
ICSI
Kulturmedium mit
Öltropfen überschichtet

Injektion eines Spermiums in eine Eizelle unter mikroskopischer Kontrolle

Selbstständiges Eindringen eines Spermiums in die Eizelle nach Zugabe von aufbreiteten Spermien

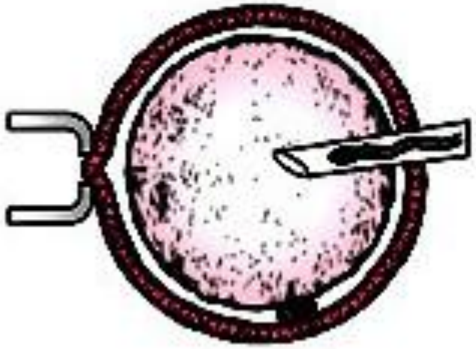
Intra cytoplasmic sperm injection (ICSI)
OLYMPUS

Intracytoplasmic Sperm Injection

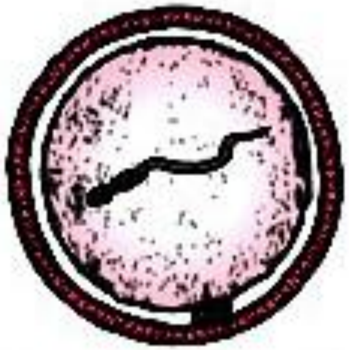


1. A micropipette 60 microns wide uses gentle suction to hold the egg.

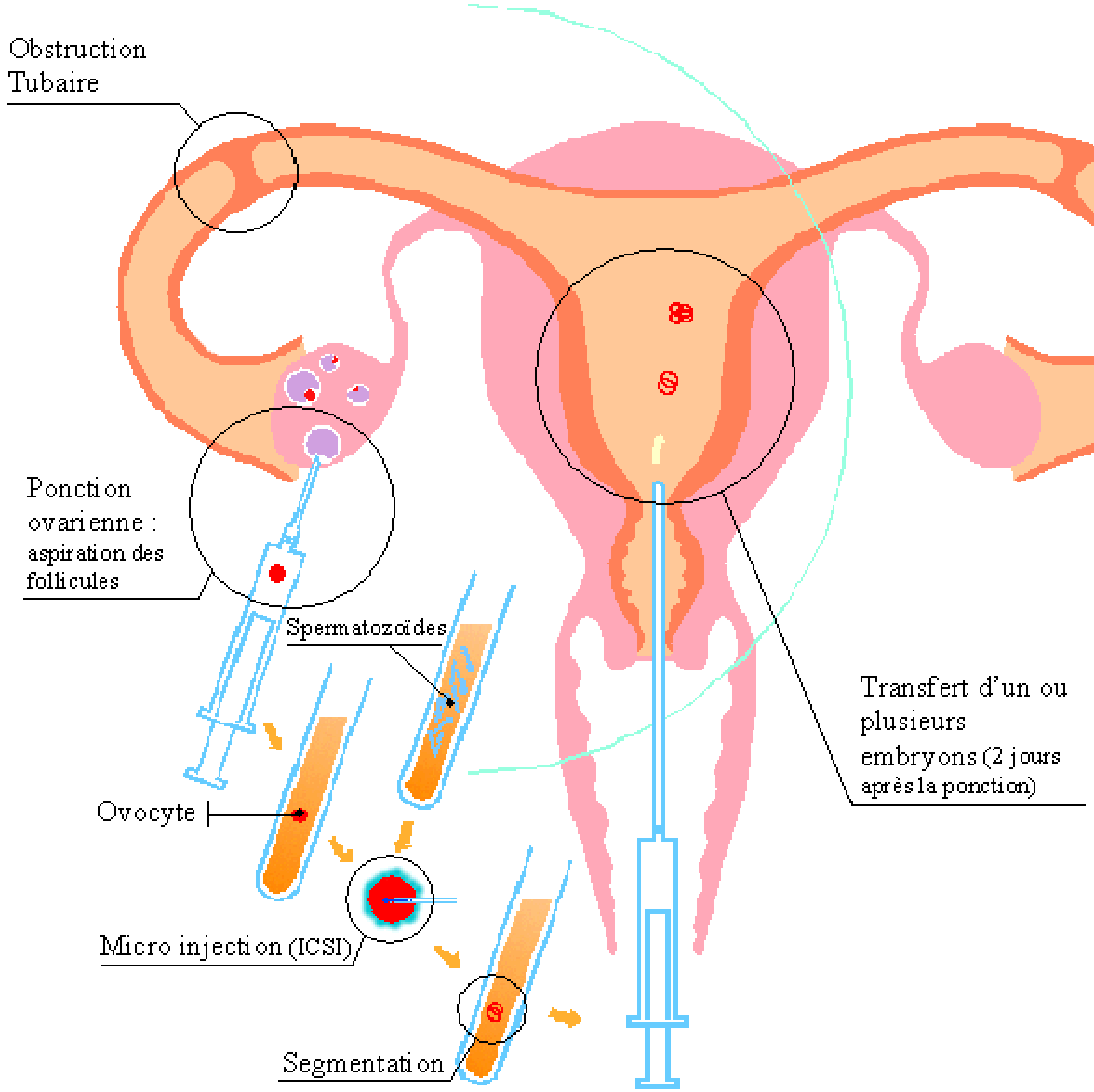
2. A single healthy sperm is taken up by an even smaller pipette 7 microns wide.

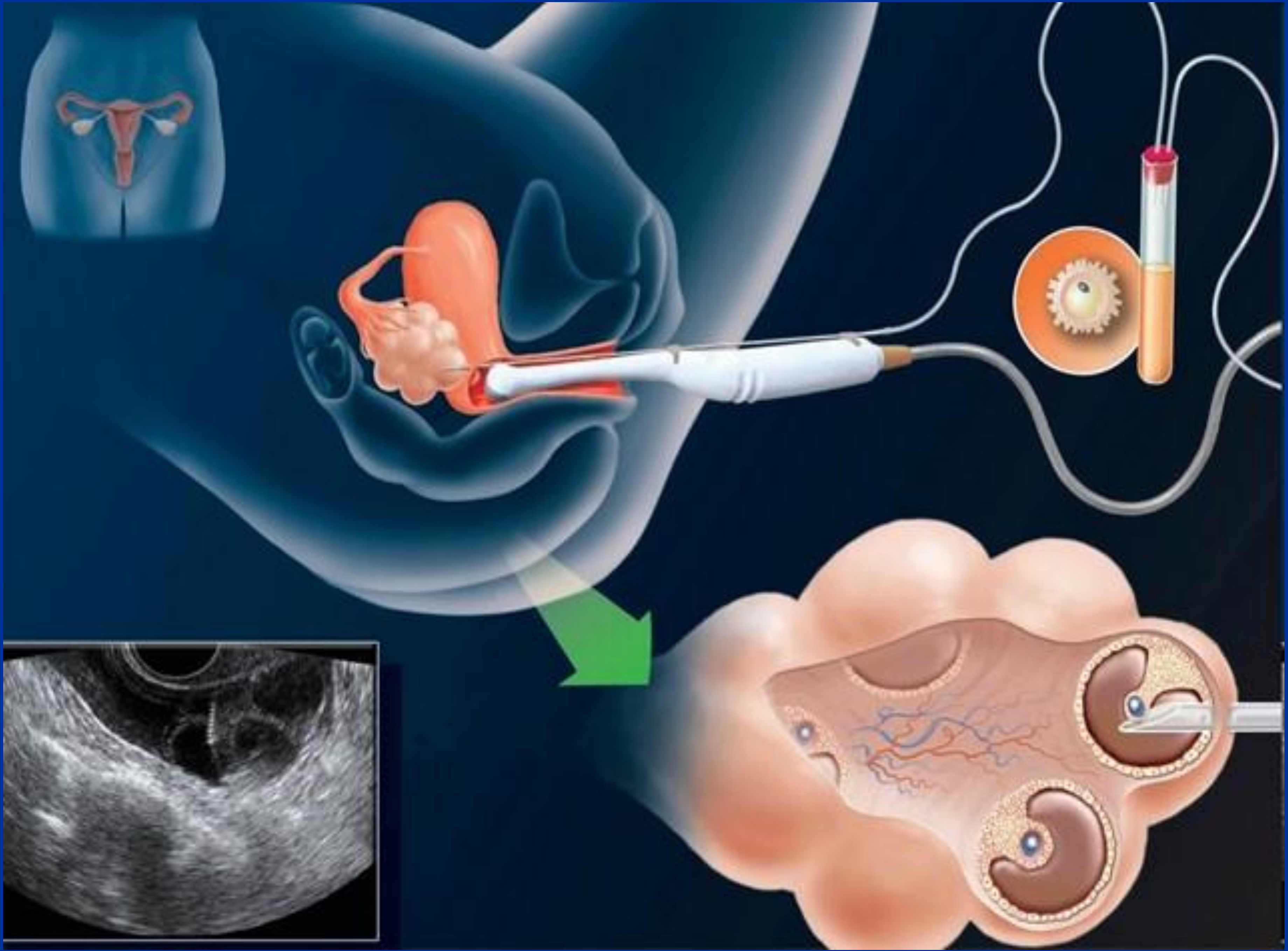


3. The pipette with the sperm is carefully inserted into the egg and the sperm is pumped out.

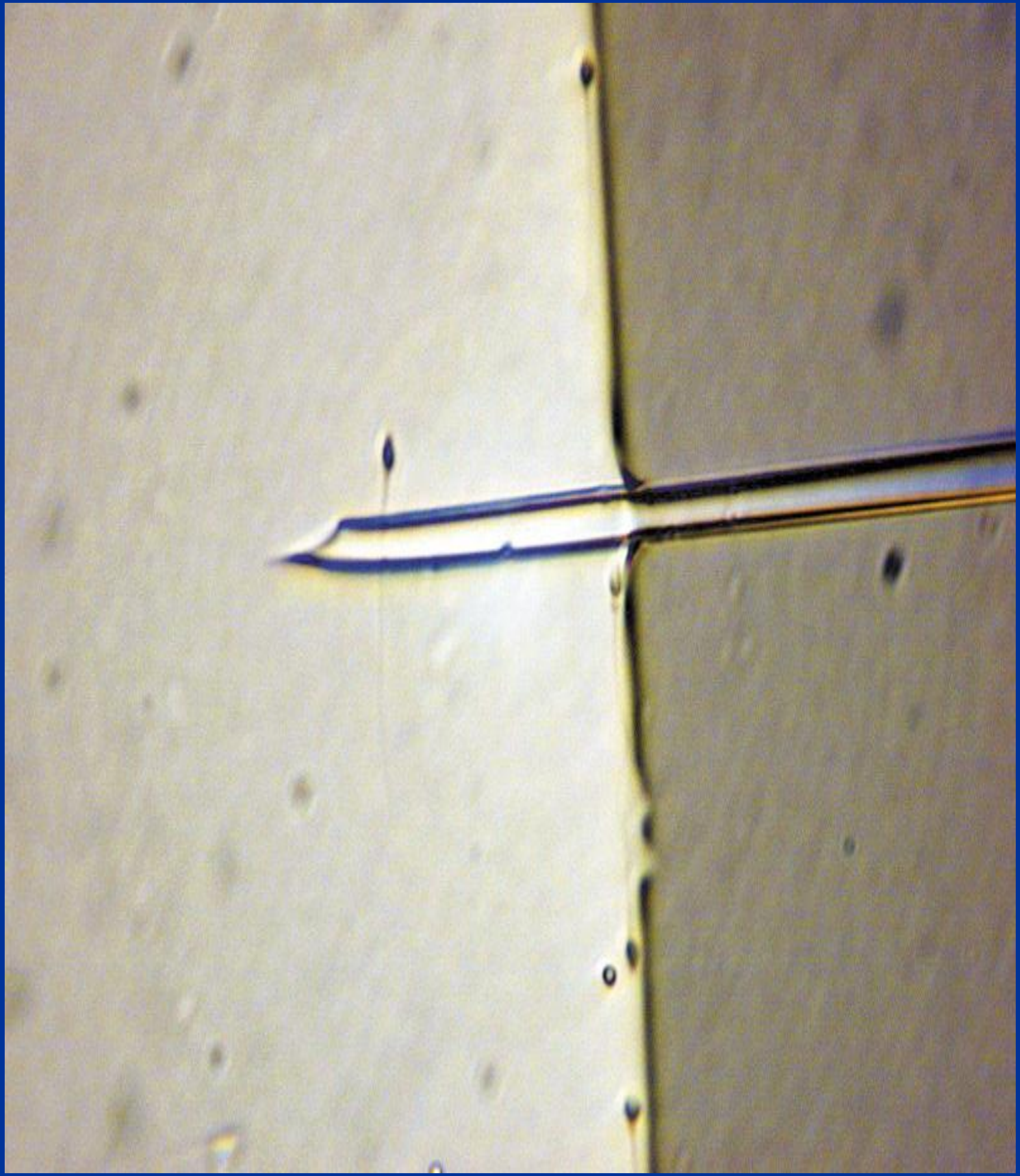


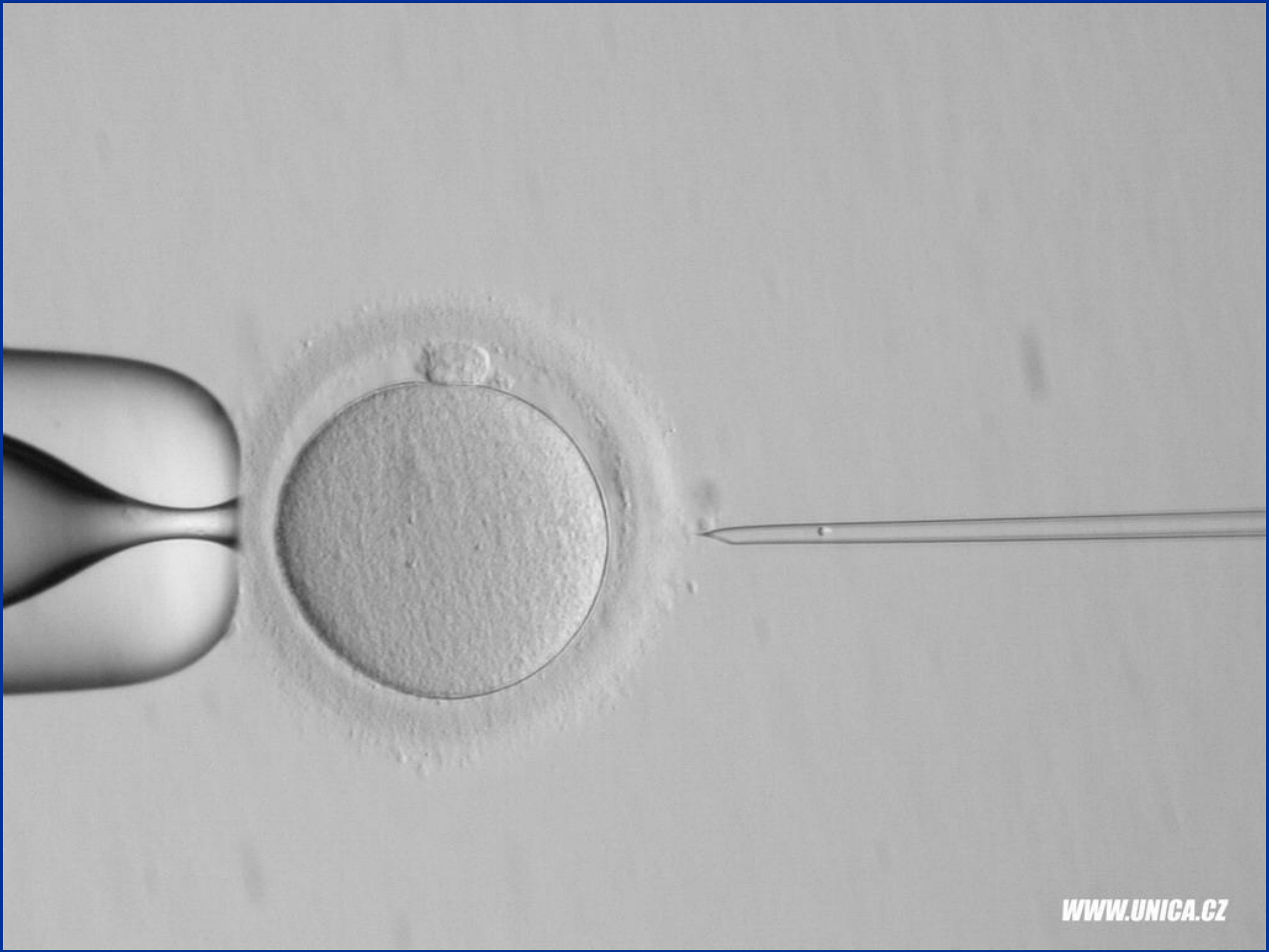
4. The pipette is withdrawn, allowing the egg to fertilize. The fertilized egg is then implanted in the woman's uterus.

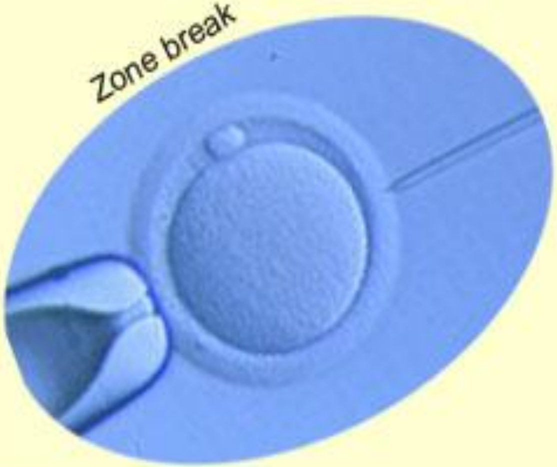

Obstruction Tubaire
Ponction ovarienne : aspiration des follicules
Spermatozoïdes
Ovocyte
Micro injection (ICSI)
Segmentation
Transfert d'un ou plusieurs embryons (2 jours après la ponction)



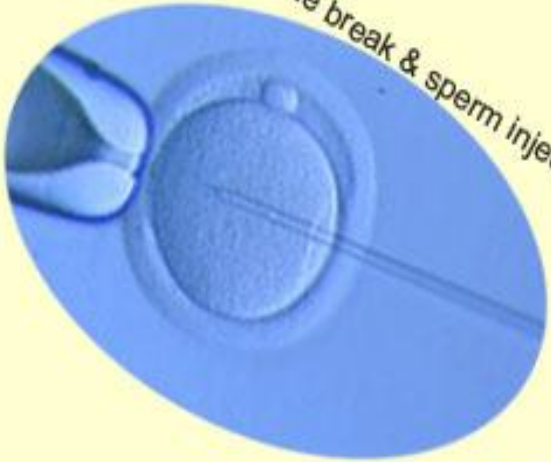
OLYMPUS
DELL



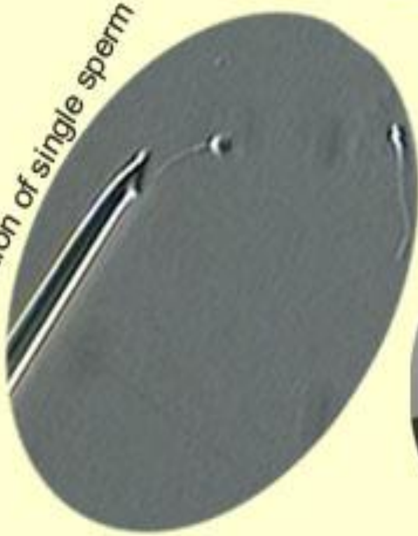

WWW.UNICA.CZ



Zone break

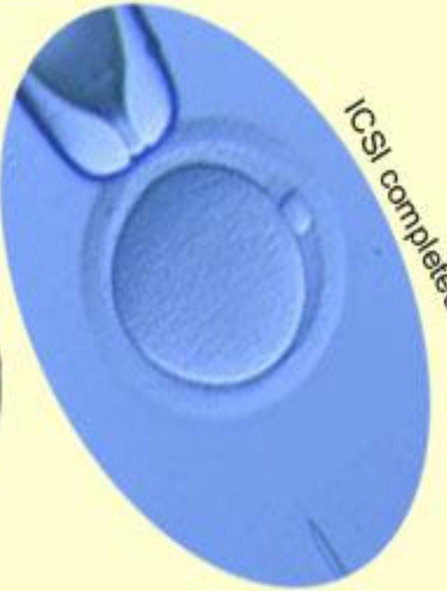


Membrane break & sperm injection



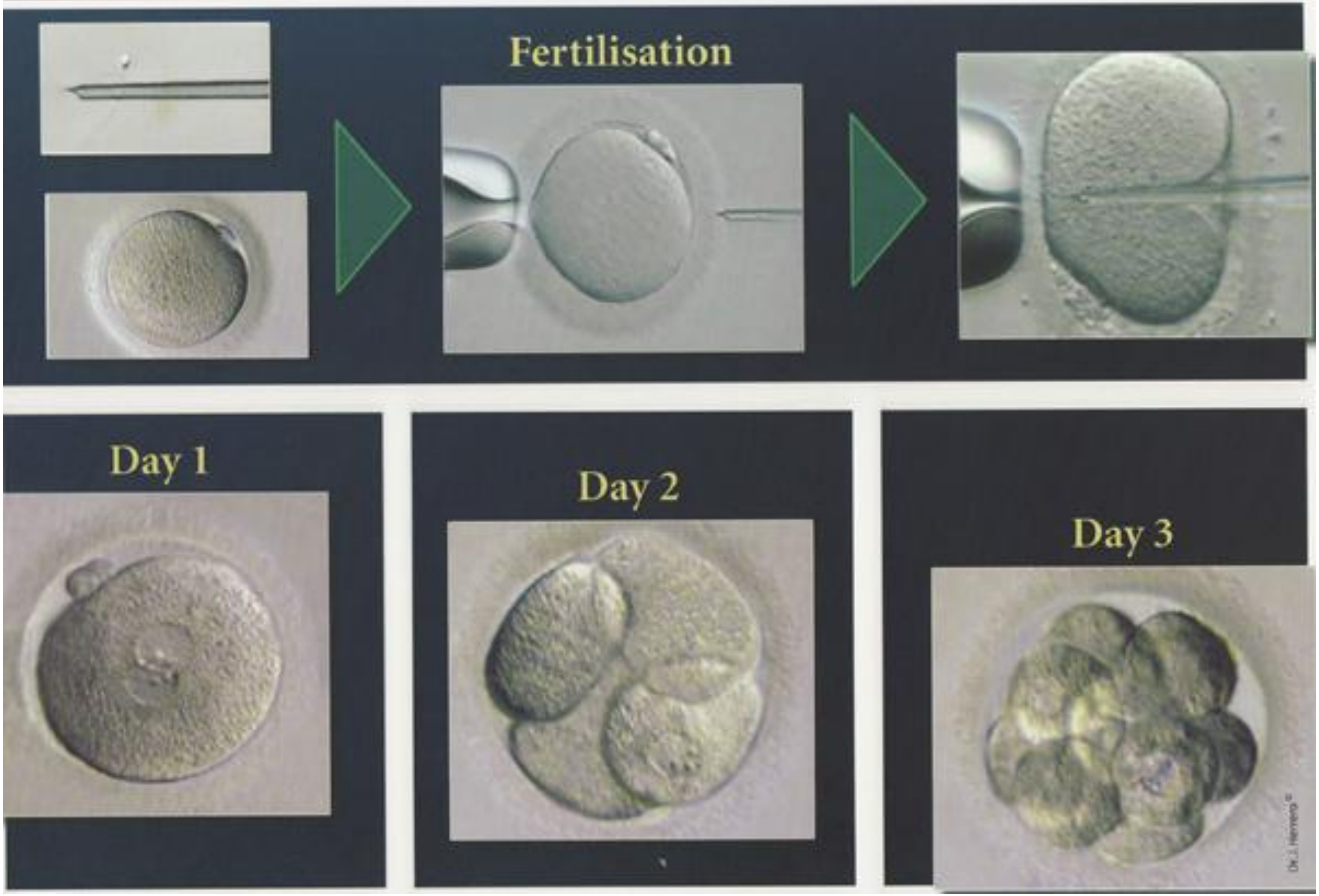
Selection of single sperm

Micro manipulator for ICSI

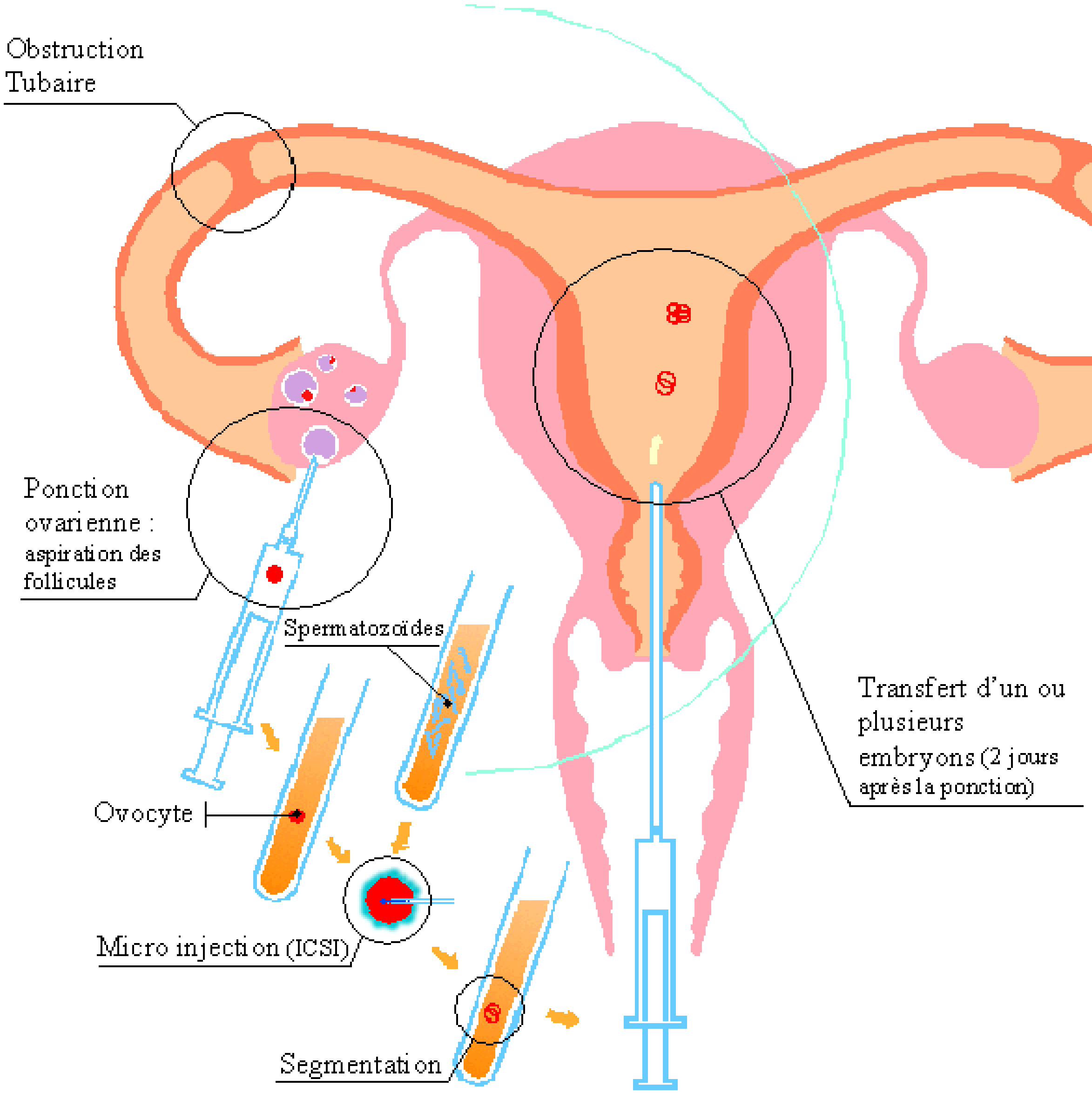


ICSI completed

ICSI and Embryo Development


Fertilisation
Day 1
Day 2
Day 3

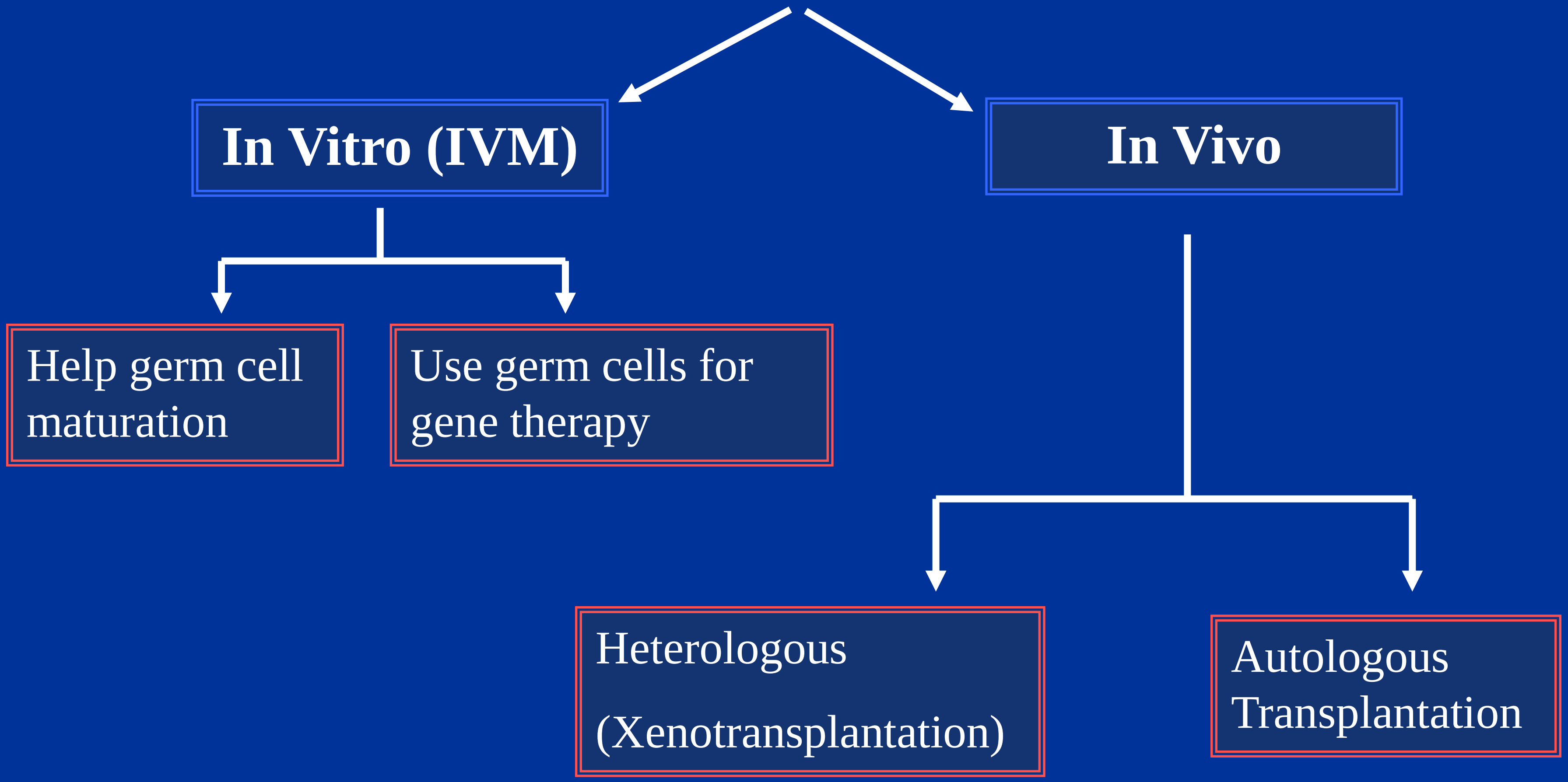
Dr J. Herrera ©


Obstruction Tubaire
Ponction ovarienne : aspiration des follicules
Spermatozoïdes
Ovocyte
Micro injection (ICSI)
Segmentation
Transfert d'un ou plusieurs embryons (2 jours après la ponction)

Future Treatment

- **Gene Therapy**: Clearly identified defects in male subfertility or sterility could be corrected by gene therapy
- **Haploidization (Reproductive semicloning):** Development of haploid nuclei from diploid somatic cells.
- **Reproductive Cloning**: Embryos formed by somatic cell nuclear transfer to enucleated oocytes (uniparental origin).
- **Stem cells:** With the absence of stem cells in the testis (eg. SCO) the generation of spermatozoa from other stem cells present in the patient body (eg. Hematopoeitic system).
- **Germ cell culture :**

Germ cell culture



In Vitro (IVM)

In Vivo

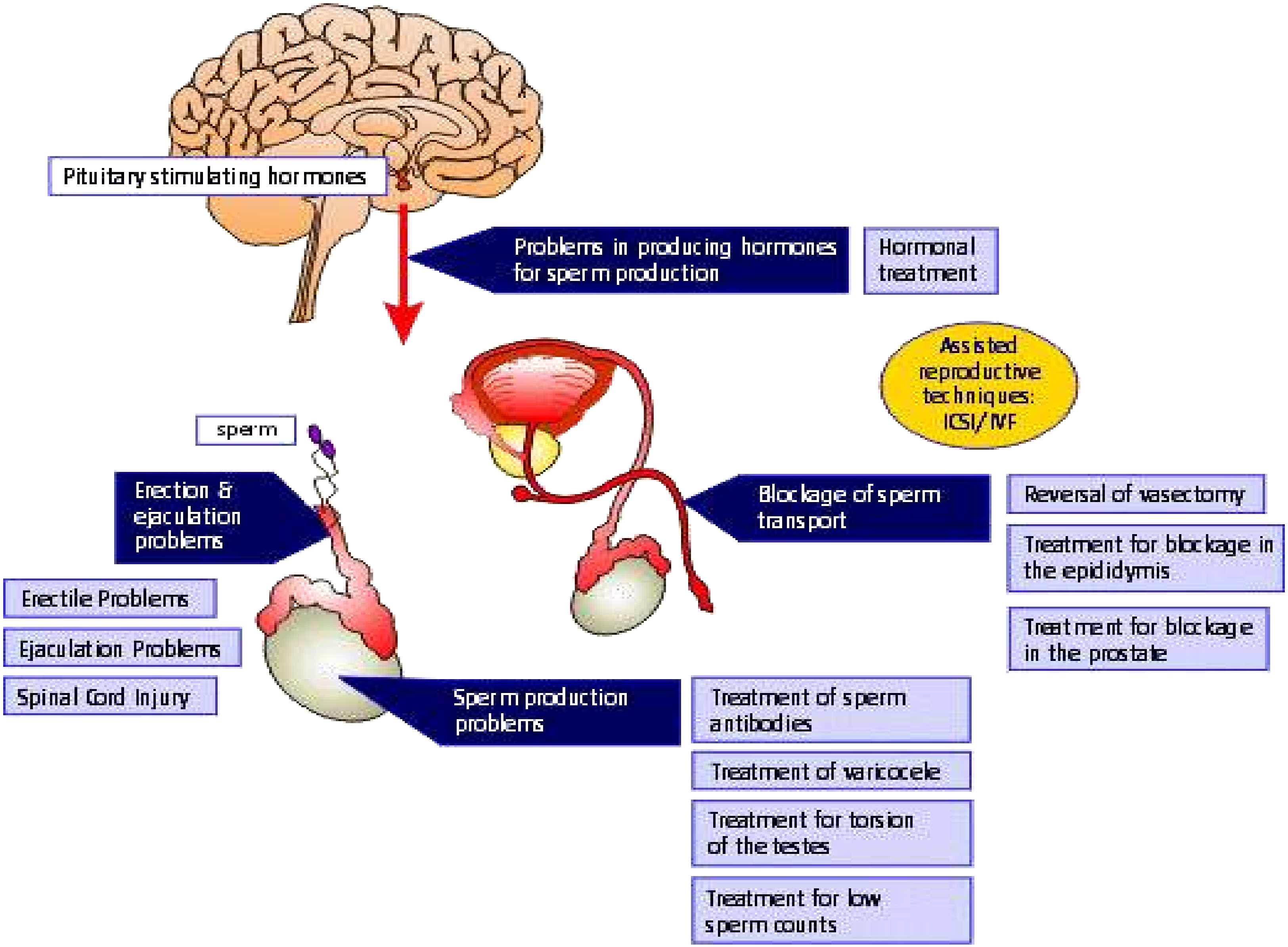
Help germ cell maturation

Use germ cells for gene therapy

Heterologous

(Xenotransplantation)

Autologous Transplantation



Pituitary stimulating hormones

Problems in producing hormones for sperm production

Hormonal treatment

Assisted reproductive techniques: ICSI/IVF

sperm

Erection & ejaculation problems

Blockage of sperm transport

Reversal of vasectomy

Treatment for blockage in the epididymis

Treatment for blockage in the prostate

Erectile Problems

Ejaculation Problems

Spinal Cord Injury

Sperm production problems

Treatment of sperm antibodies

Treatment of varicocele

Treatment for torsion of the testes

Treatment for low sperm counts

- (لله ملك السماوات والأرض يخلق ما يشاء يهب لمن يشاء اناثا ويهب لمن يشاء الذكور أو يزوجهم ذكرانا وإناثا ويجعل من يشاء عقيما انه عليم قدير). الشورى

صدق الله العظيم

GOOD LUCK

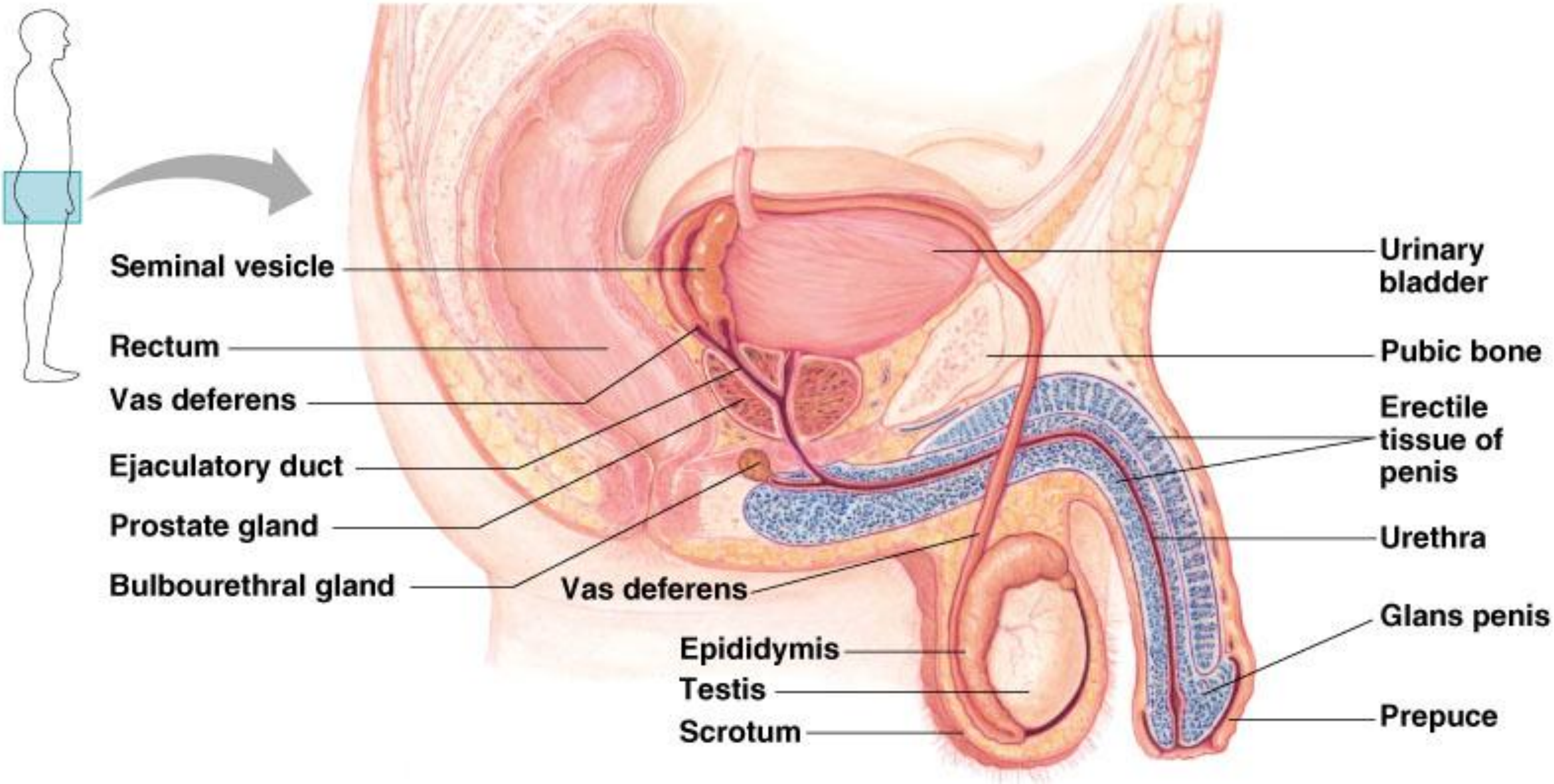
Erectile Dysfunction

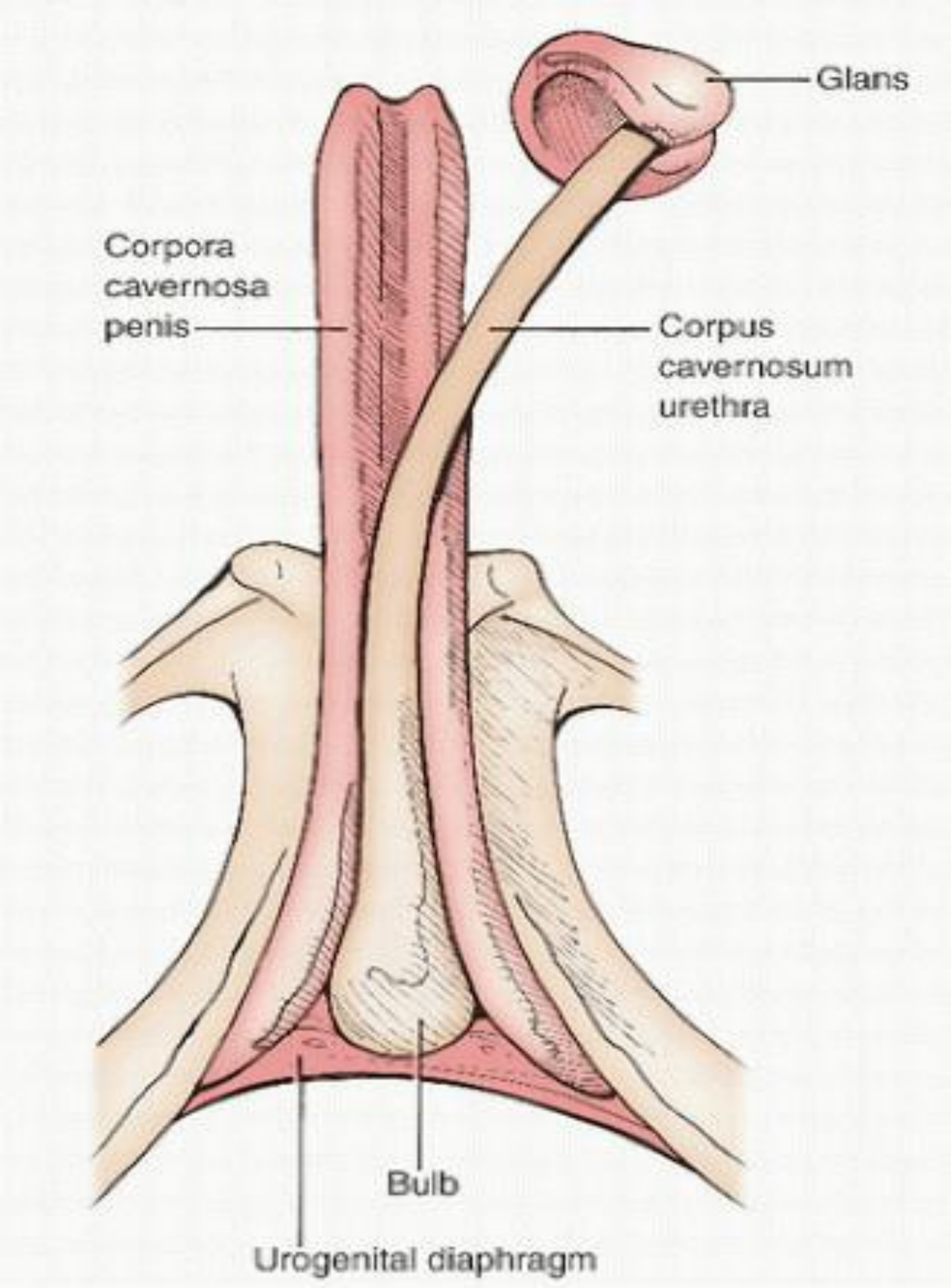
Dr. Mohammad M. Abdulgabbar
Assistant Professor of Andrology & STDs
Sana'a University

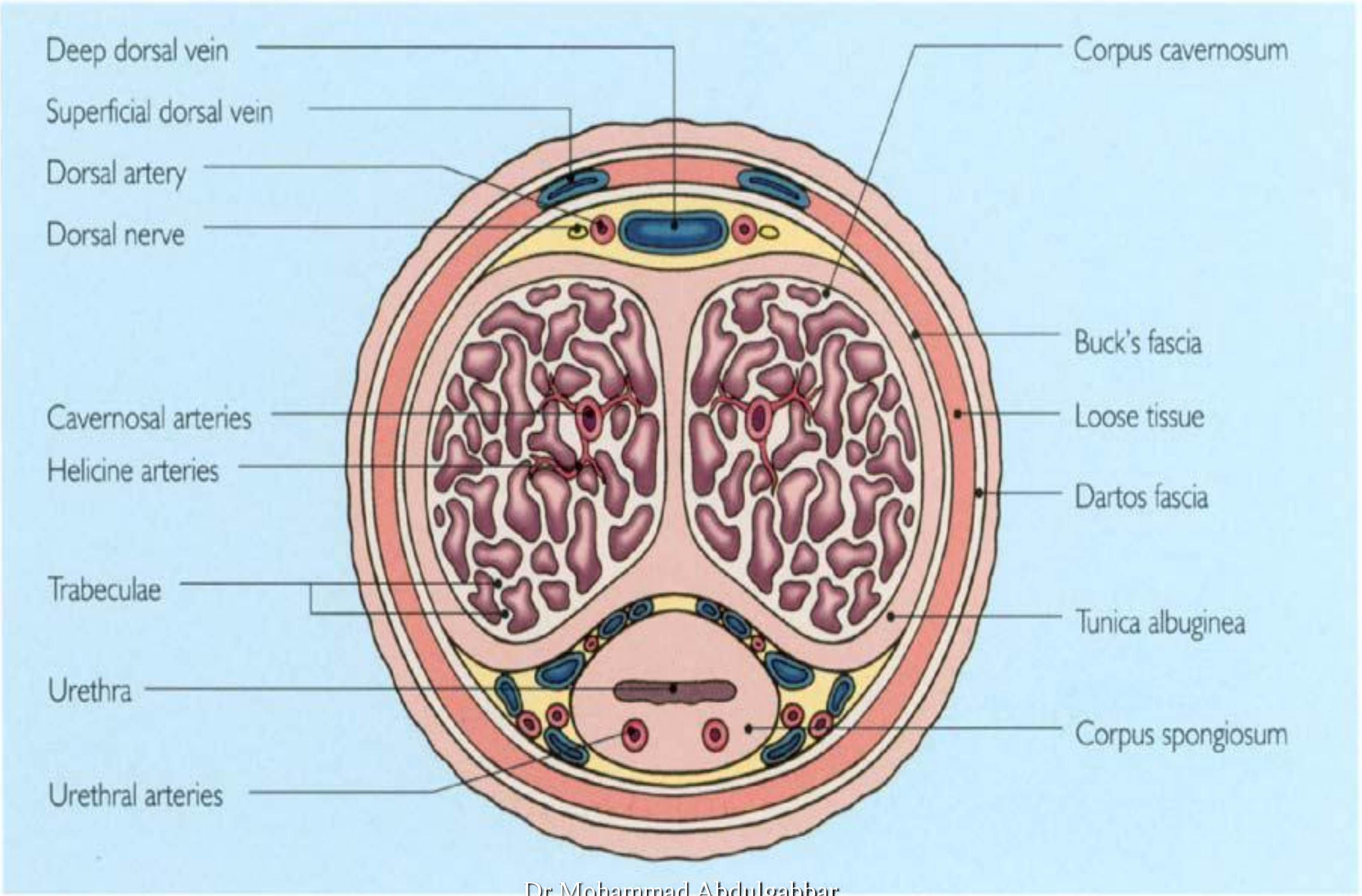
Erectile Dysfunction

- Definition
- Types
- Causes
- Diagnosis
- Treatment

Male Reproductive System

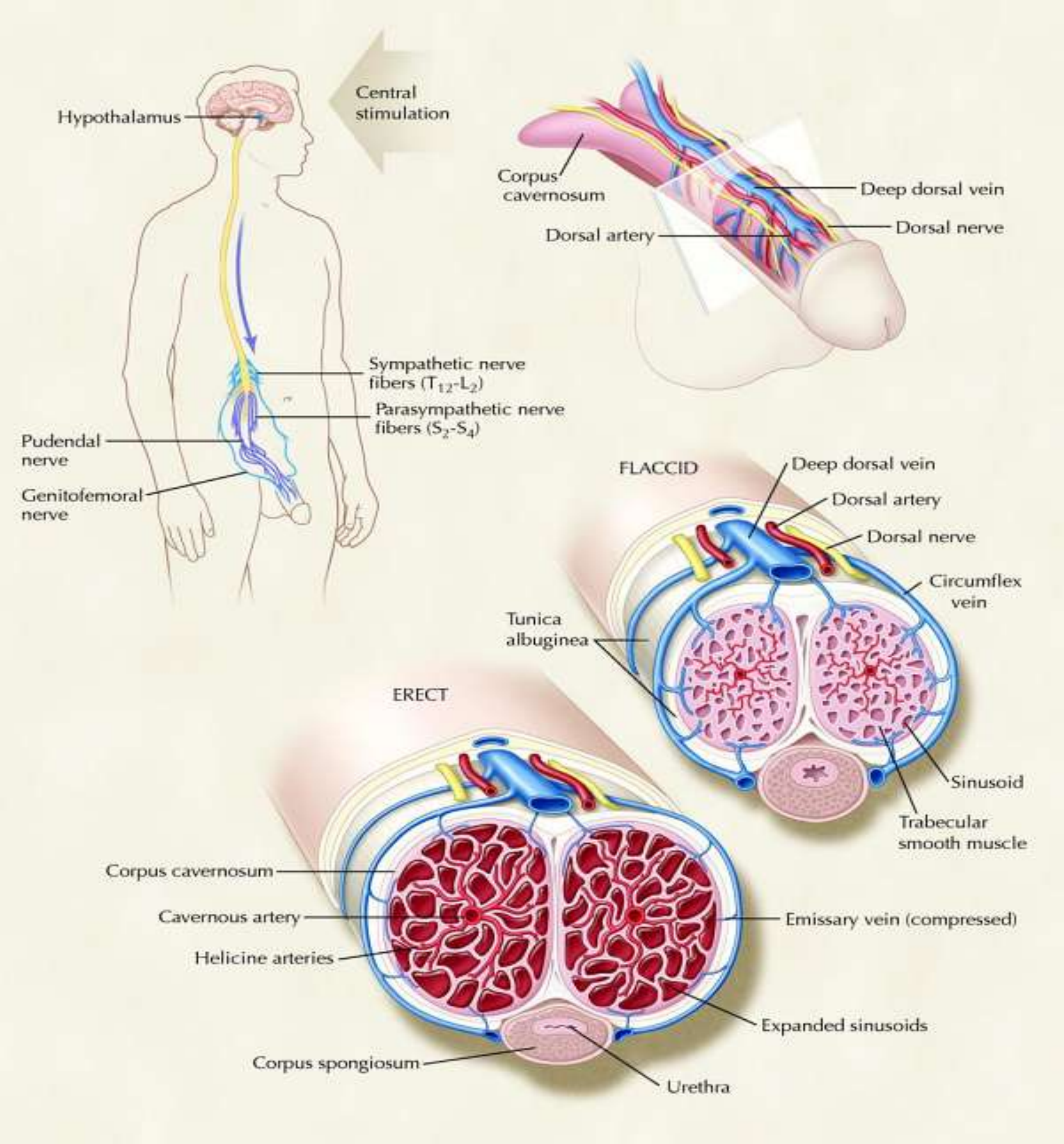

Seminal vesicle
Rectum
Vas deferens
Ejaculatory duct
Prostate gland
Bulbourethral gland
Vas deferens
Epididymis
Testis
Scrotum
Urinary bladder
Pubic bone
Erectile tissue of penis
Urethra
Glans penis
Prepuce


Glans
Corpora cavernosa penis
Corpus cavernosum urethra
Bulb
Urogenital diaphragm


Deep dorsal vein
Superficial dorsal vein
Dorsal artery
Dorsal nerve
Cavernosal arteries
Helicine arteries
Trabeculae
Urethra
Urethral arteries
Corpus cavernosum
Buck's fascia
Loose tissue
Dartos fascia
Tunica albuginea
Corpus spongiosum

Anatomy of the Penis

- **3 Coverings**: (skin, colle`s fascia, Buck`s fascia).
- **3Compartments**: (2 C.Cavernosa, 1 C. spongiosum).
- **3 Components**: (Tunica albuginea, Areolar layer of smith, Erectile (cavernous) tissue).
- **3 Arteries**: common penile artery gives 3 branches (Dorsal, Cavernous, Bulbourethral arteries).
- **3 Venous systems**: (Superficial system, Intermediate system, Deep system).
- **Muscles**: (Ischiocavernosus, Bulbospongiosus ms).
- **Ligaments**: (Fundiform, Suspensory ligs)

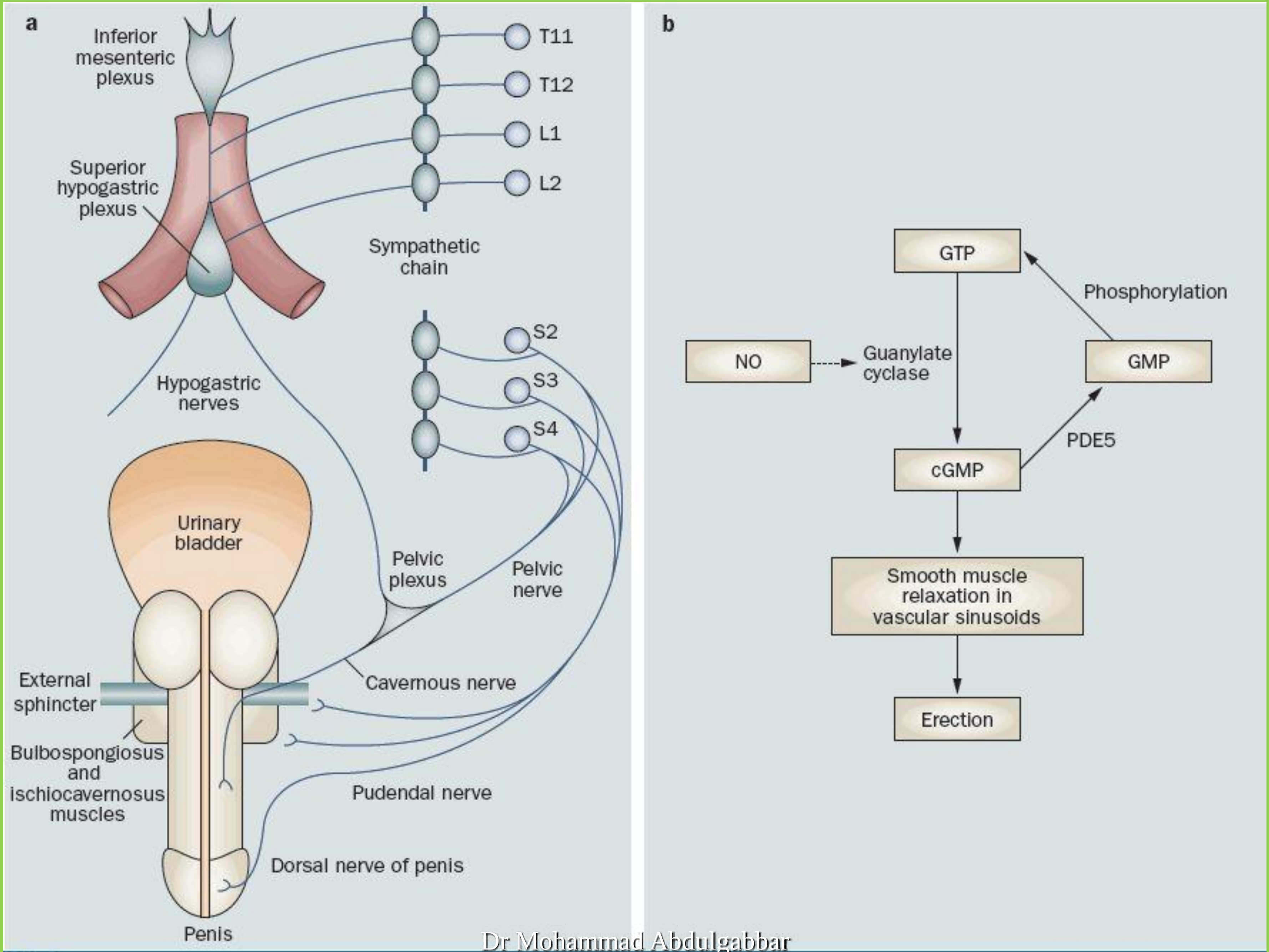

Hypothalamus
Central stimulation
Corpus cavernosum
Deep dorsal vein
Dorsal nerve
Dorsal artery
Sympathetic nerve fibers (T_{12}-L_2)
Parasympathetic nerve fibers (S_2-S_4)
Pudendal nerve
Genitofemoral nerve
FLACCID
Deep dorsal vein
Dorsal artery
Dorsal nerve
Circumflex vein
Tunica albuginea
ERECT
Sinusoid
Trabecular smooth muscle
Corpus cavernosum
Cavernous artery
Helicine arteries
Emissary vein (compressed)
Expanded sinusoids
Corpus spongiosum
Urethra

Arterial blood supply

- Cavernous arteries
- Dorsal arteries
- Bulbourethral arteries

Nerve supply

- **Autonomic pathway** (**Cavernosal** nerve):
  - **Sympathatic** (T11- L2)
  - **Parasympathatic** (S2-S4)
- **Somatic pathway** (**Pudendal** nerve):S2-S4
  - **Sensory** branch (afferent) → dorsal nerve of the penis
  - **Motor** branch (efferent) → to ischiocavernosus & bulbocavernosus muscles.

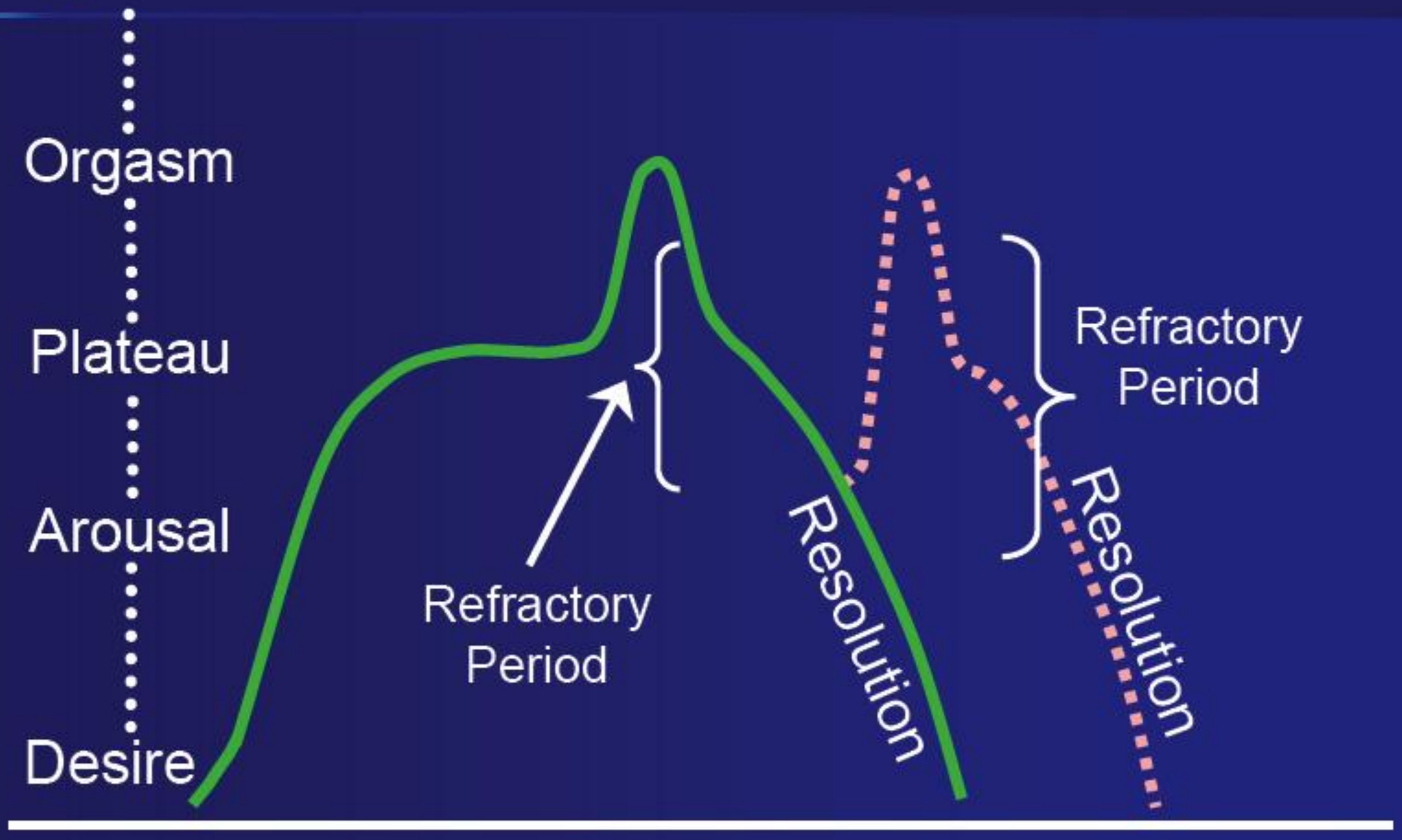

a
Inferior mesenteric plexus
Superior hypogastric plexus
T11
T12
L1
L2
Sympathetic chain
S2
S3
S4
Hypogastric nerves
Urinary bladder
Pelvic plexus
Pelvic nerve
External sphincter
Cavernous nerve
Bulbospongiosus and ischiocavernosus muscles
Pudendal nerve
Dorsal nerve of penis
Penis
b
GTP
Phosphorylation
NO
Guanylate cyclase
GMP
PDE5
cGMP
Smooth muscle relaxation in vascular sinusoids
Erection

Dr Mohammad Abdulgabbar

Medscape

Source: Nat Rev Urol © 2010 Nature Publishing Group

Male Sexual Response Cycle


Orgasm
Plateau
Arousal
Desire
Refractory Period
Resolution
Refractory Period
Resolution

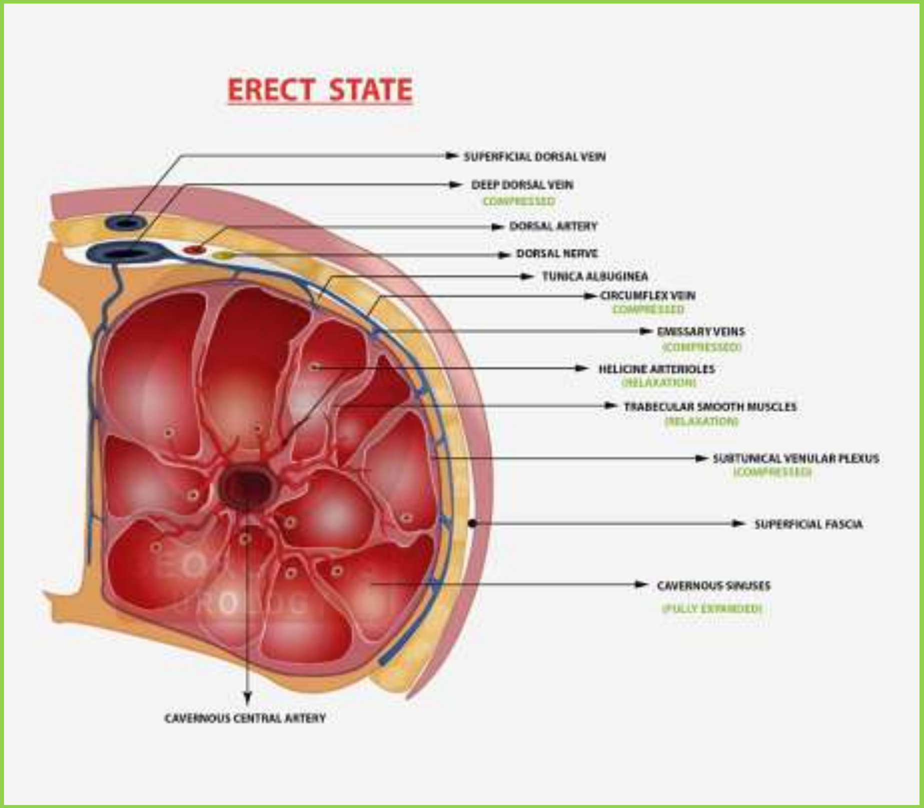
Adapted from Masters WH, Johnson VE. Human Sexual Response. Little Brown; 1966.
Adapted from Kaplan HS. *J Sex Marital Ther* 1979;3:3-9.

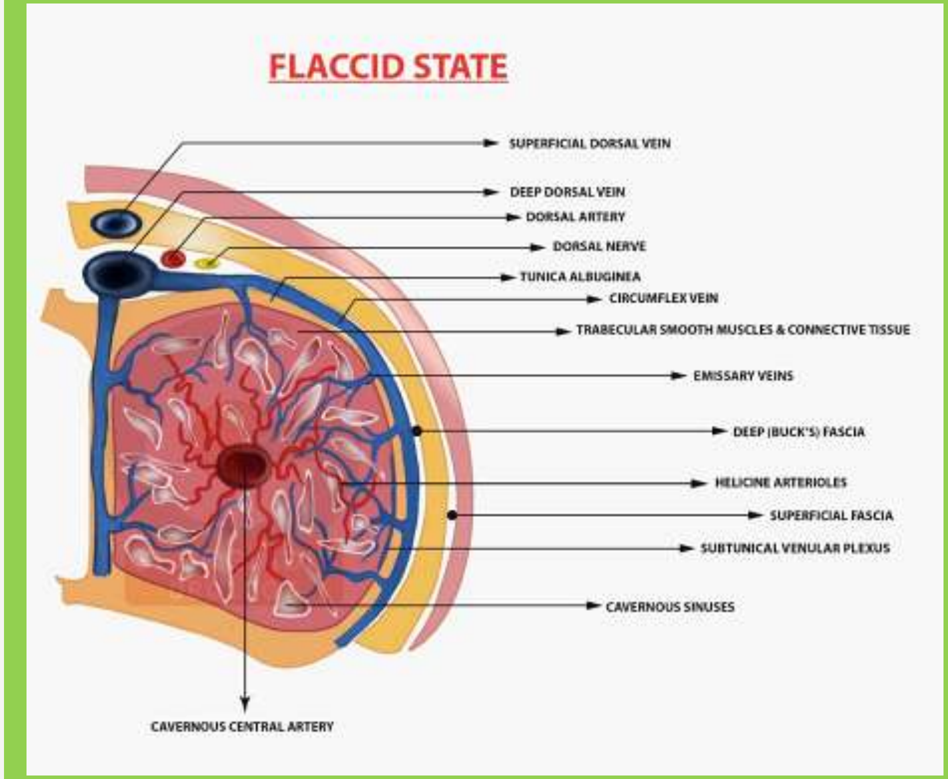
- **Psychogenic erection**
- **Reflexogenic erection**
- **Nocturnal erections**

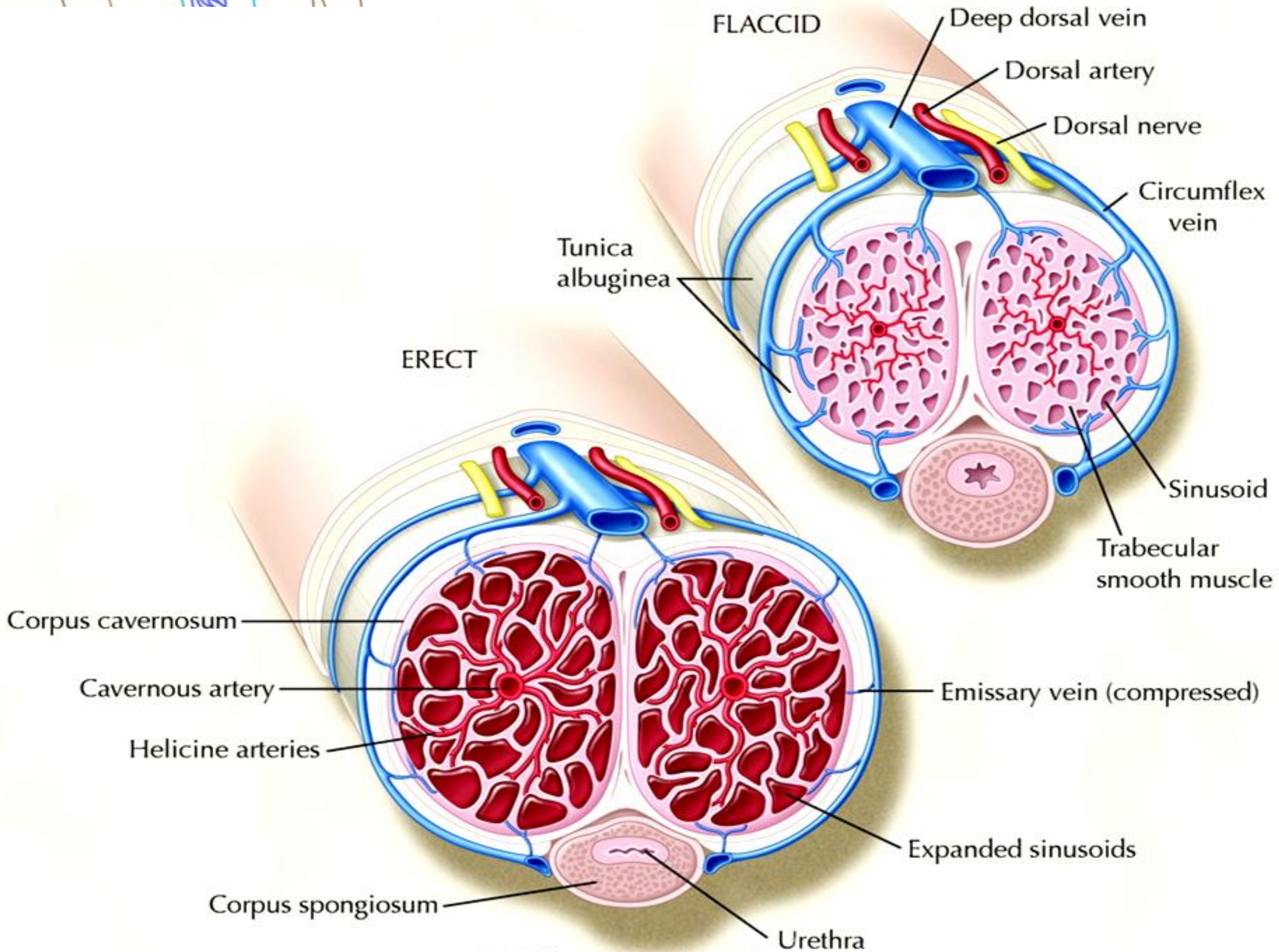
ERECTION ?

- **Erection** is a vascular phenomenon, which is mediated by neurological stimuli and influenced by hormonal factors.
- From thea neurological point, there are 2 type of erection:
  - **Psychogenic** erection (centrally stimulated or non-contact erection): If visual, auditory, olfactory, memorizing or imaginative stimuli are integrated in the limbic system as being sexual, descending tracts carry impulses from the brain modulate the spinal erection centers (T11 through L2 and S2 through S4) to activate the erectile process.
  - **Reflexogenic** erection (genital-stimulated or contact): Tactile stimulation of the genital area sends afferent impulses along the pudendal nerve, which reach the sacral segments S2-4 the efferent impulses of which are parasympathetic. This type of erection is preserved in patients who have upper spinal cord lesions.

- The neurological impulses lead to erection by mediating the following vascular response:
  - Maximum dilatation of the cavernosal artery.
  - Relaxation of smooth muscle fibers around the cavernous spaces of the corpora cavernosa.
  - Passive venous occlusion results from compression of small venules between the expanding cavernous spaces as well as the emissary and sub-tunical veins under the tunica albuginea (veno-occlusive mechanism).
  - Through this mechanism, blood is entrapped within the cavernous spaces under high pressure (~100mmHg) leading to penile lengthening and rigidity. This is augmented by the ishiocavernosus & bulbospongiosus ms → Full Rigidity.


ERECT STATE
SUPERFICIAL DORSAL VEIN
DEEP DORSAL VEIN
COMPRESSED
DORSAL ARTERY
DORSAL NERVE
TUNICA ALBUGINEA
CIRCUMFLEX VEIN
COMPRESSED
EMISSARY VEINS
(COMPRESSED)
HELICINE ARTERIOLES
(RELAXATION)
TRABECULAR SMOOTH MUSCLES
(RELAXATION)
SUBTUNICAL VENULAR PLEXUS
(COMPRESSED)
SUPERFICIAL FASCIA
CAVERNOUS SINUSES
(FULLY EXPANDED)
CAVERNOUS CENTRAL ARTERY


FLACCID STATE
SUPERFICIAL DORSAL VEIN
DEEP DORSAL VEIN
DORSAL ARTERY
DORSAL NERVE
TUNICA ALBUGINEA
CIRCUMFLEX VEIN
TRABECULAR SMOOTH MUSCLES & CONNECTIVE TISSUE
EMISSARY VEINS
DEEP (BUCK'S) FASCIA
HELICINE ARTERIOLES
SUPERFICIAL FASCIA
SUBTUNICAL VENULAR PLEXUS
CAVERNOUS SINUSES
CAVERNOUS CENTRAL ARTERY


FLACCID
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Sinusoid
Trabecular smooth muscle
ERECT
Corpus cavernosum
Cavernous artery
Helicine arteries
Emissary vein (compressed)
Expanded sinusoids
Corpus spongiosum
Urethra

Penile Erection

Neurophysiology of penile erection:

-Supraspinal pathways and centers:

- Forebrain regions (The Limbic system).
- Brainstem regions.

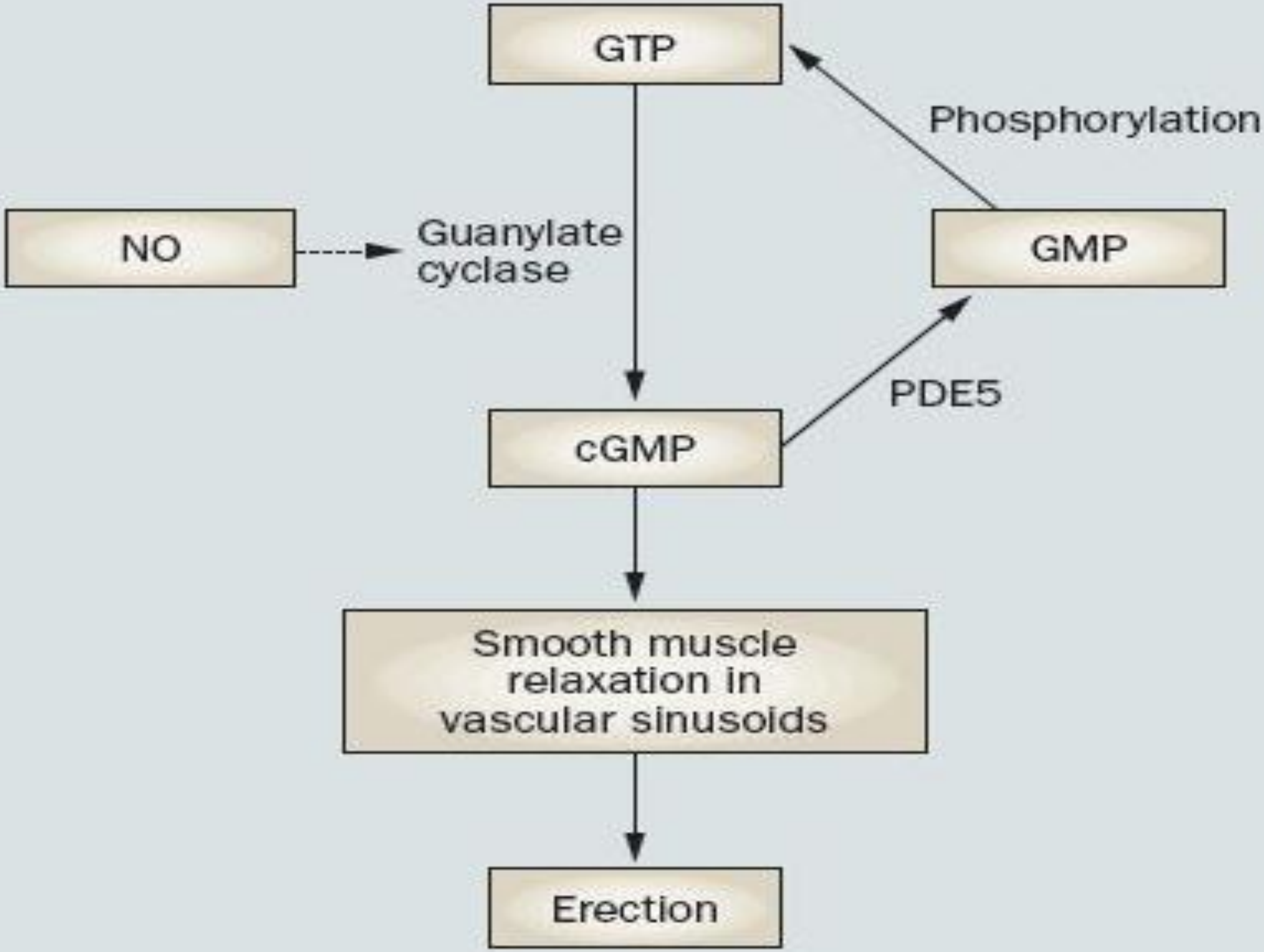
-Spinal cord and local neural pathways:

- Autonomic pathways.
- Somatic pathways.

Haemodynamic aspects:

-Vascular phase (full erection)

-Muscular phase (rigid erection)



GTP

Phosphorylation

NO

Guanylate cyclase

GMP

PDE5

cGMP

Smooth muscle relaxation in vascular sinusoids

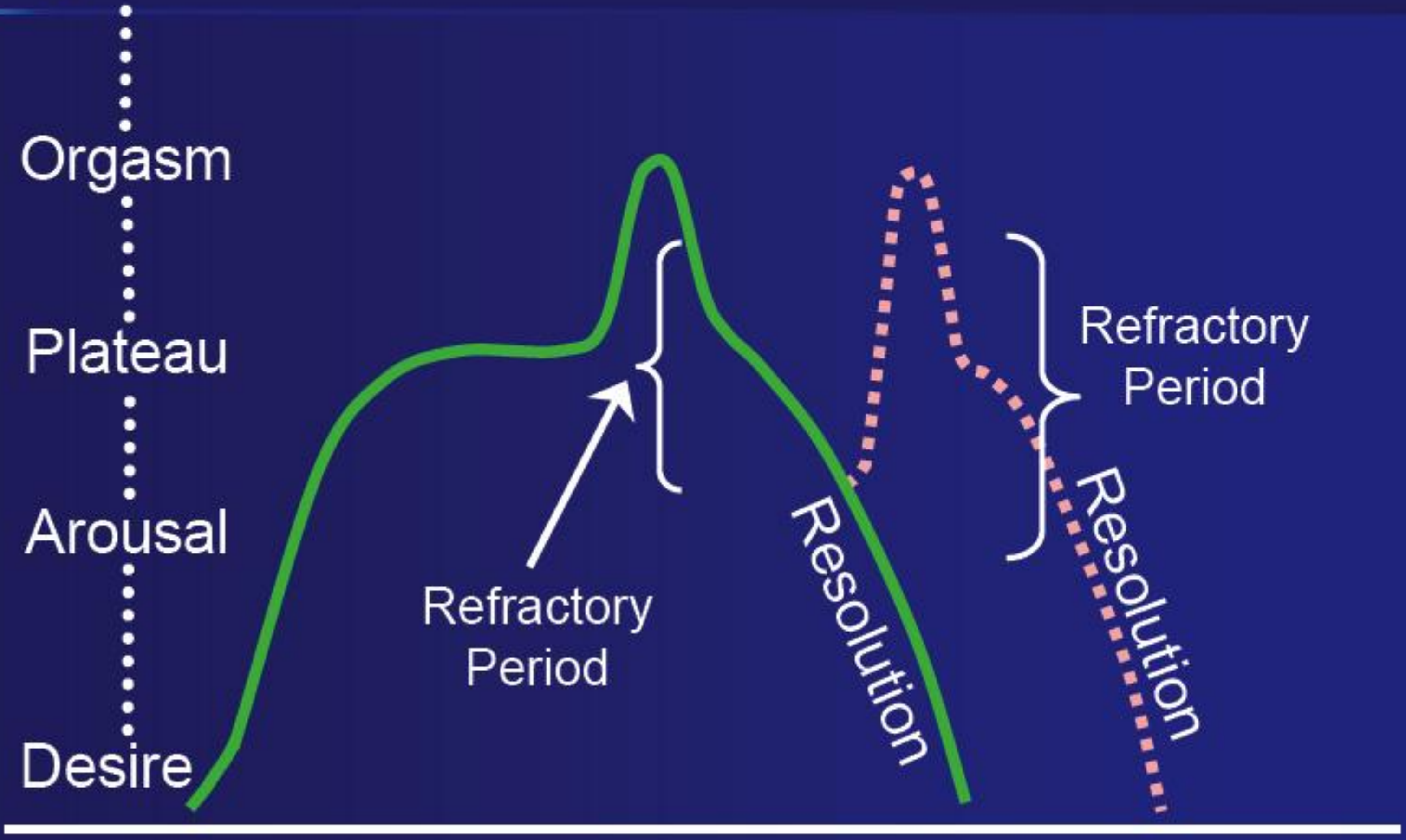
Erection

Sexual dysfunctions

- **Erectile Dysfunction** (psychogenic, organic)

- **Ejaculatory dysfunction** (Premature ejaculation, Delayed ejaculation, Retrograde ejaculation, Anejaculation…etc)

Male Sexual Response Cycle


Orgasm
Plateau
Arousal
Desire
Refractory Period
Resolution
Refractory Period
Resolution

Adapted from Masters WH, Johnson VE. Human Sexual Response. Little Brown; 1966.
Adapted from Kaplan HS. *J Sex Marital Ther* 1979;3:3-9.

Erectile dysfunction
(Impotence, Weak erection)

- **Definition**: persistent inability to **obtain** and or to **maintain** an erection sufficient for satisfactory sexual intercourse.

Etiology of ED

Etiology

- **Psychogenic**: 50% .
- **Organic**: 50% includes :
  - Endocrinal causes.
  - Medical causes.
  - Neurogenic causes
  - Vasculogenic (Arterioginic, Venogenic)
  - Penile causes
  - Drugs
- **Combined**

Psychogenic ED

Masters and Johnson's classification:

- Developmental factors
- Cognitive factors
- Neurotic factors
- Interpersonal factors

Psychogenic ED

Developmental factors:

- Maternal or Paternal dominance
- Conflicted parent-child relationship.
- Negative parents` attitude towards sex.
- Traumatic childhood sexual experience
- Traumatic first coital experience (e.g. with a prostitute)..
- Homosexuality (Female figure is not exciting)

Cognitive factors:

- Lack of sex education.
- Religious orthodoxy.
- Acceptance of cultural myths.

Neurotic factors

- Anxiety: very common cause of ED.
- Depression.
- Phobia: venerophobia – pregnophobia

Interpersonal factors:

- Poor communications: female non-cooperative, 2ry gain.
- Lack of physical attraction.
- Hostility: absence of love.
- Disgust: Female partners neglect her personal hygiene.

Organic

Endocrinal causes

- Hypothalmic disorders.
- pituitary disorders.
- Thyroid disorders
- Adrenal disorders
- Pancreatic disorders (**DM**)

Medical causes

- ED associated with systemic diseases:

  (CVS, Respiratory, Hepatic, Renal).
- ED associated with therapeutic drugs:

  (CVS drugs, CNS drugs, Digestive syst. Drugs)
- ED associated with non therapeutic drugs:

  (Tobacco, Alcohol, cocain, Khat??)

Neurogenic causes

- **Brain lesions**: cerebral accidents due to trauma, tumours or vascular accidents.
- **Spinal cord lesions:** due to trauma or other lesions such disc herniation, syringomyelia.
- **Peripheral nerves lesions**: pelvic fracture or TURP, prostatectomy.

Vasculogenic ED

1. Arteriogenic causes:

- **Nontraumatic**:Hypercholesterolemia, Hyperglycemia, Hypertension, Smoking, Aging.
- **Traumatic**: pelvic or perineal vessels injury during operations or non iatrogenic causes as with bicycle riders.

2. Corporovenous causes:

- Type 1= Congenital abnormal venous drainage (Primary ED) ⟶ **Reversible**
- Type 2= Abnormal tunica albuginea (aging, DM, Peyronie`s disease).
- Type 3= Abnormal cavernous smooth muscle (fibrosis, degeneration)
- Type 4= Inadequate release of the neurotransmitters.
- Type 5= Acquired abnormal venous drainage as after shunt operation for priapism.⟶ **Reversible**

Penile causes

- **Peyronie`s disease**: fibrotic plaque develops within the tunica albuginia of corpora cavernosa.
- **Neglected Priapism** (ischemic) :prolonged painful penile erection (not sexual) lasts more than 4 hours and fail to subside even after orgasm.
- **Untreated fracture penis.**

- **Risk factors of ED**
  - Hypertension
  - Hyperglycemia
  - Hyperlipidemia
  - Habits (**smoking**, alcohol, cocain….Qat??)
  - Ageing, **obesity**, life style….

DIAGNOSIS OF ED

Diagnosis of ED

1. **History** : personal history – present history – sexual history – past history – medical history – psychiatric history – partner history
2. **Examination** : General & Local
3. **Investigation** (Diagnostic procedures for ED)

Differentiation between Psychogenic and Organic

	Psychogenic	**Organic**
Onset	Acute	Gradual
Circumstances	Situational	Global
Course	Variable	Constant, progressive
Non coital erection (ME, NE)	Rigid	Poor
Psychosocial problems	Long history	Secondary
Anxiety	Primary	Secondary
Risk factors	Not present	Maybe present

Examination

General :

- Manifestations of hypogonadism .
- Manifestations of systemic & endocrinal diseases (e.g. anemia, hepatic, renal, cardiac diseases, hypo-hyperthyroidisms..etc
- Vascular examination (BP, Peripheral pulses).
- Neurological examination (sensations & reflexes on lower limbs (e.g., cauda equina lesion, paraplagia…)

Local:

- Penis : size , induration , fibrosis , curvature.
- Testes : site, size , consistency .
- DRE (PR): Anal sphincter tone, prostate.
- Genital reflexes: Cremasteric, scrotal reflex, Bulbocavernosus reflex.

Investigation (Diagnostic procedures for ED)

- **Laboratory**: mandatory tests (Blood sugar, serum T, Prolactin…..Lipids), others accordingly.
- **RigiScan**
- Penile vascular studies: **ICI**, **pharmacopenile duplex** ultrasonography (testing arteriogenic ED), cavernosometry & cavernosography (testing Venogenic ED).
- Neurological evaluation: Genital reflexes, Biothesiometry.

Treatment of ED

- Management of the cause
- Sex therapy
- Non-surgical treatment (Oral, ICI, Topical, VCD)
- Surgical (e.g., Penile prosthesis)
- Future ttt of ED (Gene therapy, tissue engineering, nerve reconstruction, stem cells)

Management of the cause

- Psychiatric treatment for deep psychic ED.
- Quit smoking, alcohol, addiction, Khat ??
- Control DM, HTN, Lipids, etc…
- Testosterone or gonadotropins for hypogonadism.
- Anti-hyperprolactemic drugs for hyperprolactinemia.

Sex Therapy

Masters and Johnson's principles of sex therapy:

- Sex does not only mean intercourse. Apart from reproductive purposes, other forms of sexual relations can be exciting and satisfying.
- Sex is not something that a man does to a woman but it is something that a man and a woman do together .
- Many cultural ideas and attitudes to sex are misleading.

Basic techniques (for all sexual dysfunctions):

- Couple therapy.
- Cotherapy (male & female therapists).
- Coordinated therapy (Andrologist & Psychiatrist).
- Starting sessions (history and examination)
- Sexual guiding and education sessions.
- Sensate focus sessions (gradual sexual exposure).

Non-surgical treatment

Oral Therapy:

- **PDEIs** (Sildenafil citrate, Vardenafil, Tadalafil (absolutely contraindicated with nitrates)
- Adrenergic receptor antagonist (yohimbine, trazadone….)
- Dopamine receptor agonist (Apomorphine)
- Testosterone (Check the prostate for cancer? PSA).

Local Therapy:

- ICI (Papaverine, Phentolamine, PGE1, Trimix, Quadmix). Diagnostic & Therapeutic.
- Intraurethral agents (MUSE, LLPGE1)
- Transdermal/Transglanular corporal drug delivery (Minoxidil, PGE1, Papaverine gel)

VCD

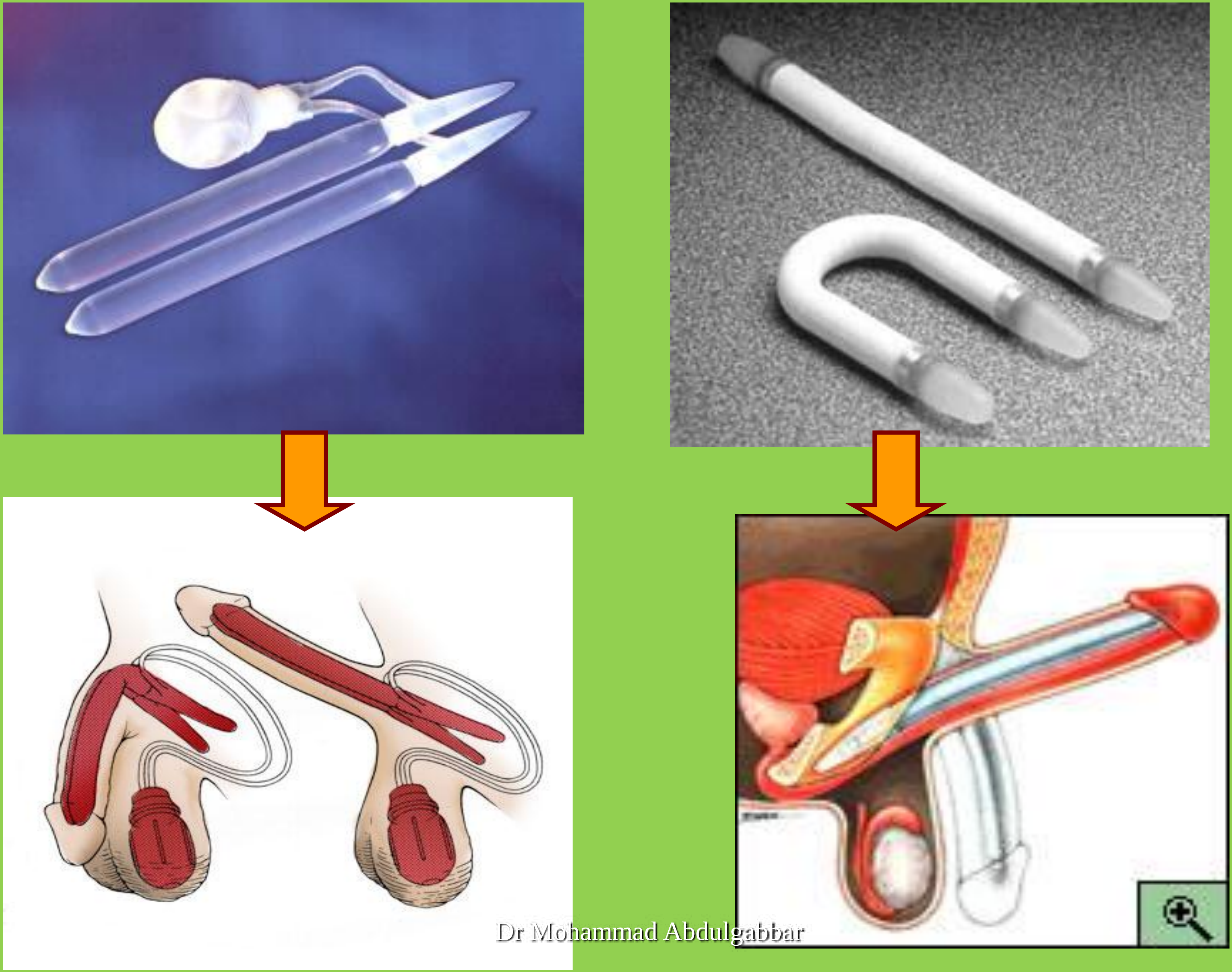
Surgical

Vascular surgery:

- Arterial revascularization.
- Venoligation ???? .

Penile prosthesis:

- Insertion of prosthesis in the corpora cavernosa. Types:
  - **Semirigid** (malleable).
  - **Inflatable**



Ejaculation

(The most common sexual disorder in men)

Dr Mohammad Abdulgabbar

- Affects up to 30% of males.
- Affects up to 75% of men at some stage during their sexual life.

Prevalence

30%

of males.
(<10% with IELT < 1min)

75%

of men at some stage during their sexual life

50%

PE and **ED** comorbid

The Three Stages of Normal Antegrade Ejaculation:

- **Emission**: Contractions of the wall of the prostate, seminal vesicles, epididymis, and vas deferens expel their contents into the posterior urethra (sympathetic T10-L 2,).
- **Ejection**: Semen is expelled from the posterior urethra to the outside after bladder neck closure & rhythmic contractions of bulbocavernous/pelvic floor muscles.(parasympathetic and somatic, S2-S4).
- **Orgasm**: Build-up and release of pressure in posterior urethra. (Smooth muscle contraction of accessory sexual organs and urethral bulb Sensation due to cerebral processing of pudendal nerve sensory stimuli).

Criteria to define PE

TIME
(IELT)

CONTROL

IMPACT

Definition of Premature Ejaculation

Masters and Johnson, 1970

Inability of a man to delay ejaculation long enough for his **partner** to reach **orgasm** on **50%** of intercourse attempts.

- A persistent or recurrent pattern of ejaculation occurring during partnered sexual activity within approximately 1 minute following vaginal penetration and before the individual wishes it … The symptom … must have been present for at least 6 months and must be experienced on almost all or all (approximately 75–100%) occasions of sexual activity (in identified situational contexts or, if generalized, in all contexts). The symptoms … cause clinically significant distress in the individual” and “The sexual dysfunction is not better explained by a nonsexual mental disorder or as a consequence of severe relationship distress or other significant stressors and is not attributable to the effects of a substance/medication or another medical condition (**DSM-V, 2013**).

- A male **sexual dysfunction** characterized by ejaculation which **always** or **nearly always** occurs **prior** to or **within** about **one minute** of vaginal penetration from the first sexual experience (Lifelong PE) or a clinically significant and bothersome reduction in latency time, often to about 3 minutes or less (acquired PE),
- the **inability** to **delay** ejaculation on **all** or **nearly all** vaginal penetrations,
- associated with **negative personal consequences**, such as distress, bother, frustration and/or the avoidance of sexual intimacy (**ISSM, 2014**).

ISSM, 2014

- ✓ **≤1 mins (Life-long),**
- ✓ **≤ 3mins (Acquired),**
- ✓ **Inability** to **delay Ejac,**
- ✓ **Negative personal consequences.**

- The inability to control ejaculation sufficiently for both partners to enjoy sexual interaction (ICD-10, 2016)

- (International statistical Classification of Diseases and related Health problems 10th revision)

Types of PE

- ☐ Life long (Primary) PE
- ☐ Acquired (secondary) PE
- ☐ Variable PE
- ☐ Subjective (premature-like) PE

PE variant	Possible etiology	Treatment
Lifelong PE (< 1min)	Neuro-biological, genetic	Pharmacotherapy, Behavioral therapy, Psychotherapy, (treat the cause)
Acquired PE (< 3min)	Medical (ED,prostatitis, hyperthyroidism..), psychological and interpersonal causes	
Subjective (premature-like) PE, <6min	Normal variant of sexual function	Psycho-education (reassurance) Behavioral therapy, Psychotherapy
Natural variable PE	Psychological	

Analysis of the C/O (diagnostic criteria)

TIME

(IELT), estimated

CONTROL

Comorbidity

(ED)

IMPACT

Type

Frequency

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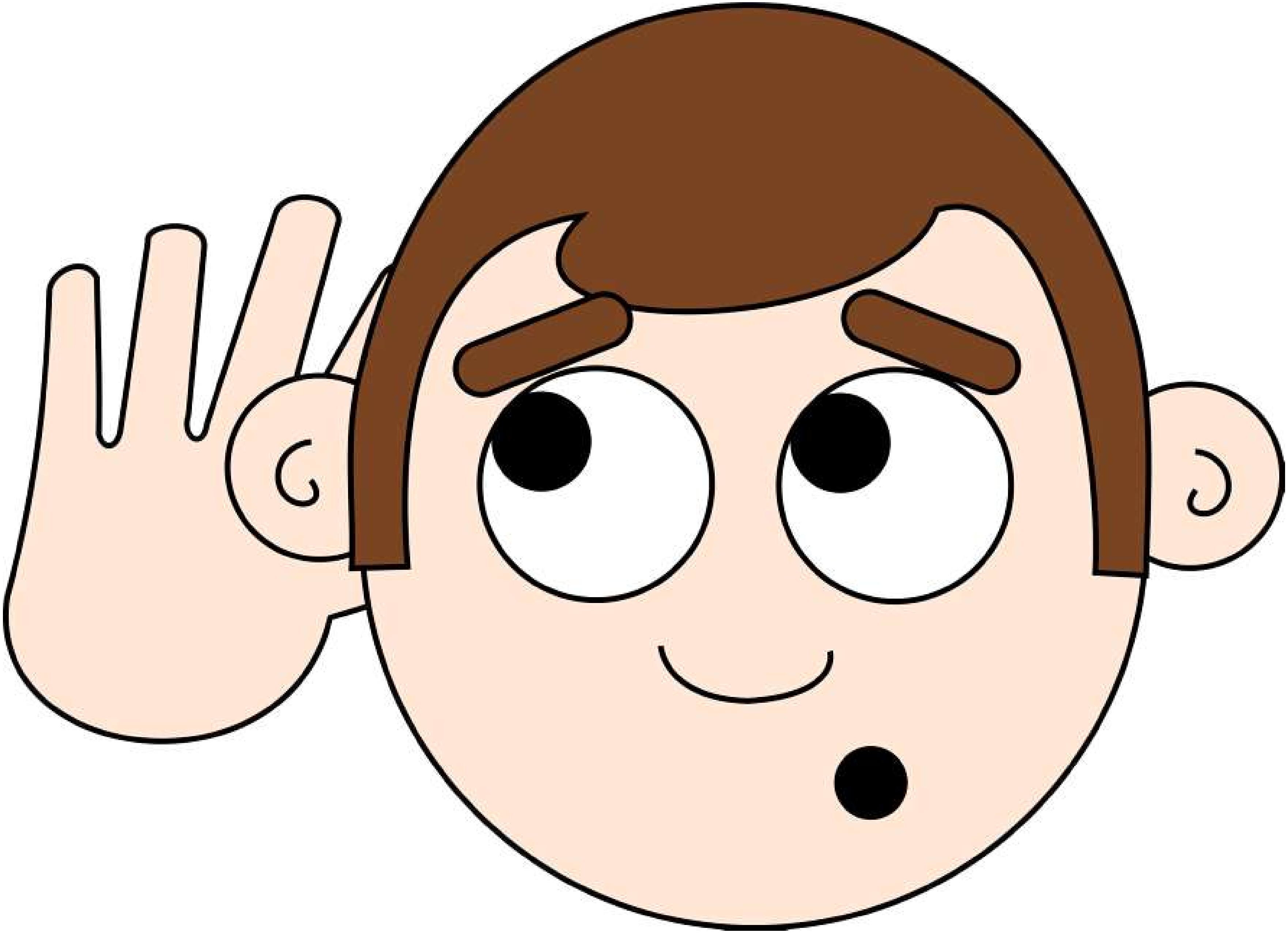

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Etiology of PE

- **Psychogenic causes** (>90%).
  - Conditioned prematurity: This occurs when the early sexual experiences have been with prostitutes, through petting or chronic heavy masturbation.
  - Subconscious hatred towards females.
  - Anxiety and over concern about partner satisfaction.
  - Unresolved marital problems.
- **Biological causes** (<10%)
  - Genetic predesposition.
  - Chronic pelvic congestion e.g. chronic prostatitis
  - Hyperthyroidism.
  - Erectile dysfunction.
  - Drug-induced e.g. sympathomimetics
  - Neurological disorders e.g. hypersensitive glans penis, MS and neuropathies.

Treatment

Drugs

Local

Sex therapy

Combined

Others

Drugs

SSRIs

(Dapoxitine)

The SSRIs remain the drugs of choice. All SSRIs have **equal** efficacy

(AUA)

Local

Local Anaesthetics

Condoms

Fortacin®
150 mg/ml
+ 50 mg/ml
cutaneous spray, solution
lidocaine/prilocaine
Cutaneous use.
5 ml
Fortacin®
Recordati Ireland Ltd.
EU/1/13/881/002

Sex therapy

psychotherapy

Behavioral

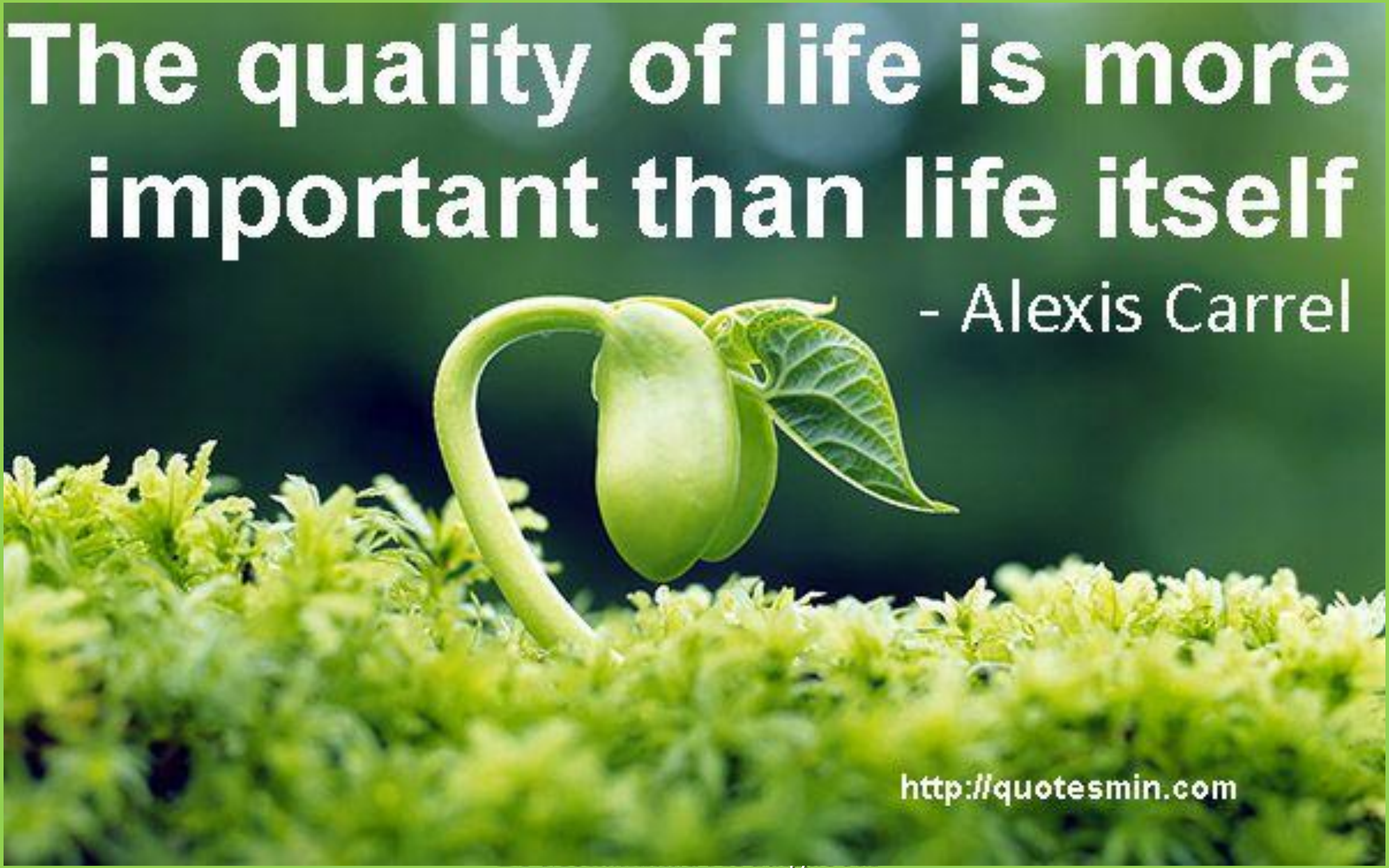
Combined

Fortacin
150 mg/ml
+ 50 mg/ml
cutaneous spray, solution
lidocaine/prilocaine
Cutaneous use.
5 ml

Impact of PE

COMPLICATIONS

Impaired overall quality of life (M/F)



The quality of life is more important than life itself

- Alexis Carrel

http://quotesmin.com

Sexual dysfunction

PE is a very adrenaline-inducing event

ED

Infertility

Psychological problems

Anxiety
(35%)

causal
?
effect

Treatment of PE

1-Minimizing penile receptivity:

-Condom

-Local anesthetics.

-Distractive thinking.

2- Sex therapy.

-Stop-squeeze technique: Wife stimulates the penis then squeezes the glans firmly when the husband is about to ejaculate. The cycle is repeated several times.

-Start-stop technique: Stimulation is stopped before ejaculation is inevitable and then resumed once more.

3- Drugs:

-SSRIs (selective serotonin reuptake inhibitors).

-Clomipramine (Anafranil)

- Dapoxetine

- Hyaluronic acid gel glans augmentation
- Botulinum toxins injections into ejaculatory muscles
- Dorsal penile nerve cryoablation

Thank You

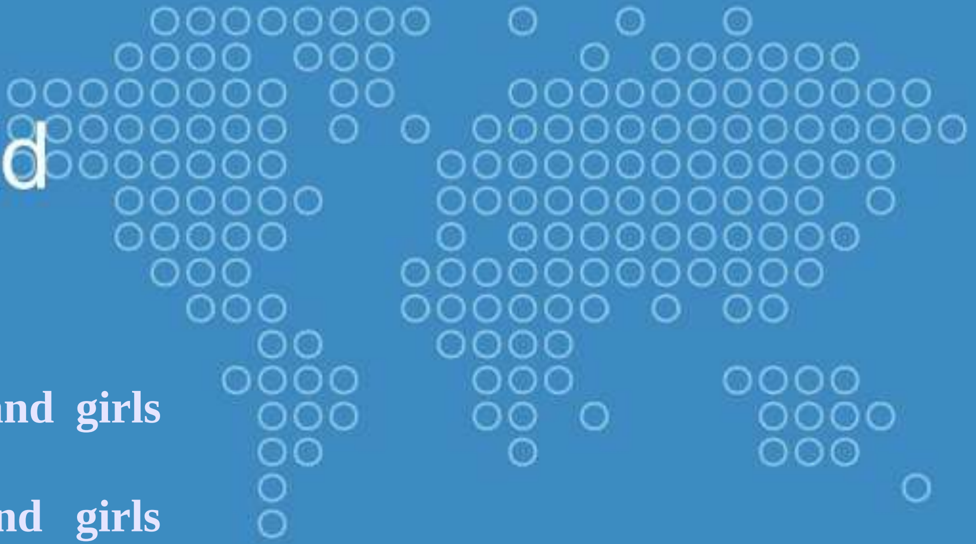
Human Immunodeficiency Virus (HIV)

By
Dr. Mohammad M. Abdulgabbar
Assist. Professor of Andrology & STDs
Sana'a University

HIV
AIDS

37.7 million
people in the world

were living with HIV in 2020.



An estimated **20.6 million** women and girls were living with HIV .
In **sub-Saharan Africa**, women and girls accounted for **63%** of all new HIV infections in 2021

AIDS

DECEMBER 1ST
WORLD
AIDS
DAY

AIDS Def.

- A term which applies to the most advanced stages of HIV infection. It is defined by the occurrence of any of more than 20 **opportunistic infections** or HIV-related **cancers**.

OR

- Their CD4 cell count drops **below 200** cells/cmm.

- **WHO (1986)** has developed a clinical case definition of AIDS that is suitable for the African and developing countries with lack of laboratory facilities as follows:

- **Major signs**

1. **Weight** loss of more than 10% of body weight.
2. Chronic **diarrhea** for more than one month.
3. Prolonged **fever** for more than one month.

- **Minor signs**

1. Persistent **cough** for more than one month.
2. **Oropharyngeal candidiasis**.
3. Recurrent herpes **zoster**.
4. Chronic progressive and disseminated herpes **simplex**.
5. Generalized pruritic **dermatosis**.
6. Generalised **lymphadenopathy**.

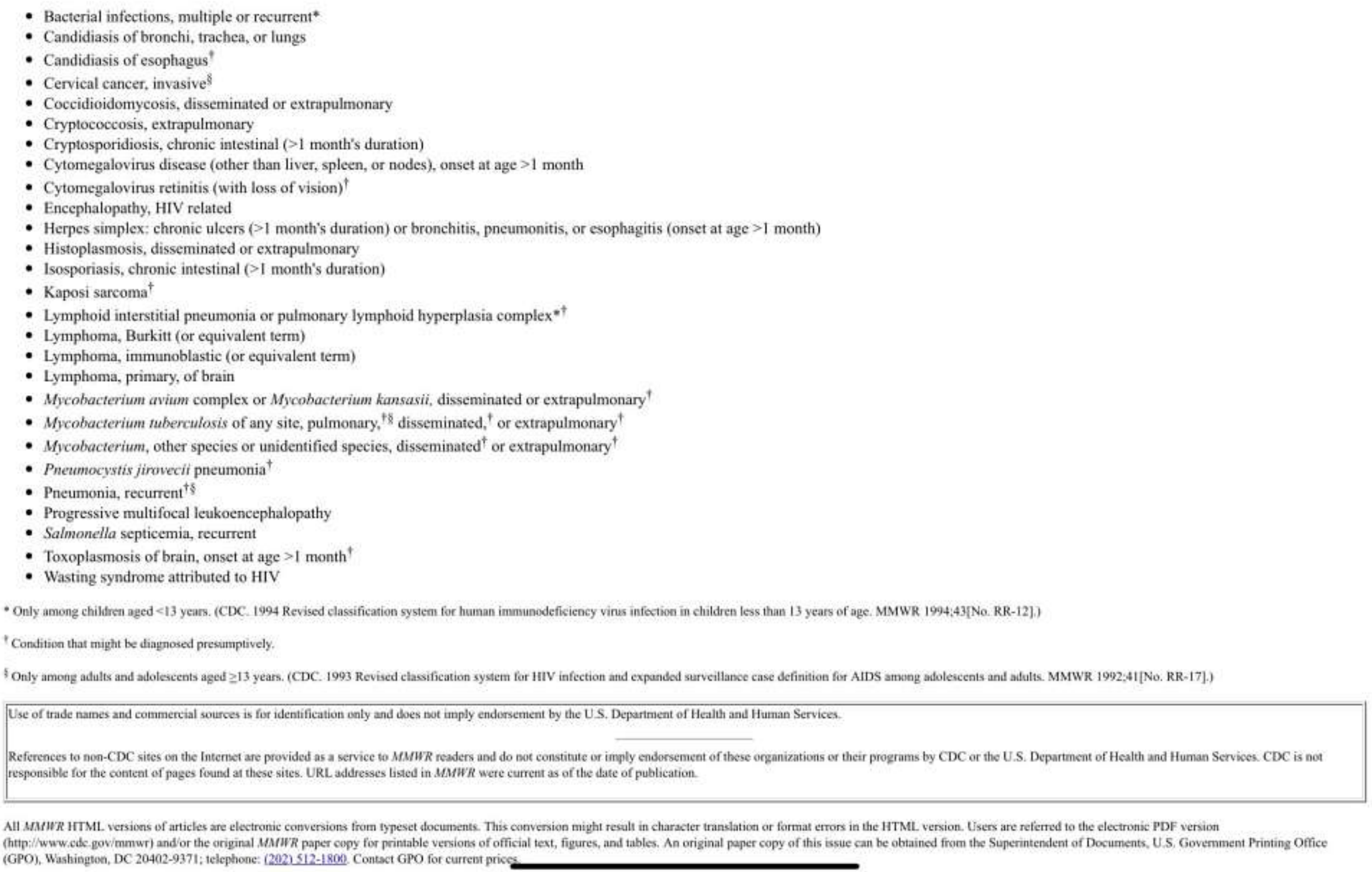
ADULTS

AIDS = at least 2 of the major signs + at least 1 of the minor signs in the absence of a known cause of immunosuppression as cancer or severe malnutrition.

CDC Definition

a CD4+ T-cell count below 200 cells/µL
a CD4+ T-cell percentage of total lymphocytes
of less than 14%
or one of the defining illnesses.

AIDS-Defining Conditions



- Bacterial infections, multiple or recurrent[*]
- Candidiasis of bronchi, trachea, or lungs
- Candidiasis of esophagus[†]
- Cervical cancer, invasive[§]
- Coccidioidomycosis, disseminated or extrapulmonary
- Cryptococcosis, extrapulmonary
- Cryptosporidiosis, chronic intestinal (>1 month's duration)
- Cytomegalovirus disease (other than liver, spleen, or nodes), onset at age >1 month
- Cytomegalovirus retinitis (with loss of vision)[†]
- Encephalopathy, HIV related
- Herpes simplex: chronic ulcers (>1 month's duration) or bronchitis, pneumonitis, or esophagitis (onset at age >1 month)
- Histoplasmosis, disseminated or extrapulmonary
- Isosporiasis, chronic intestinal (>1 month's duration)
- Kaposi sarcoma[†]
- Lymphoid interstitial pneumonia or pulmonary lymphoid hyperplasia complex[*†]
- Lymphoma, Burkitt (or equivalent term)
- Lymphoma, immunoblastic (or equivalent term)
- Lymphoma, primary, of brain
- *Mycobacterium avium* complex or *Mycobacterium kansasii,* disseminated or extrapulmonary[†]
- *Mycobacterium tuberculosis* of any site, pulmonary,[†§] disseminated,[†] or extrapulmonary[†]
- *Mycobacterium*, other species or unidentified species, disseminated[†] or extrapulmonary[†]
- *Pneumocystis jirovecii* pneumonia[†]
- Pneumonia, recurrent[†§]
- Progressive multifocal leukoencephalopathy
- *Salmonella* septicemia, recurrent
- Toxoplasmosis of brain, onset at age >1 month[†]
- Wasting syndrome attributed to HIV

[*] Only among children aged <13 years. (CDC. 1994 Revised classification system for human immunodeficiency virus infection in children less than 13 years of age. MMWR 1994;43[No. RR-12].)

[†] Condition that might be diagnosed presumptively.

[§] Only among adults and adolescents aged ≥13 years. (CDC. 1993 Revised classification system for HIV infection and expanded surveillance case definition for AIDS among adolescents and adults. MMWR 1992;41[No. RR-17].)

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2014 CDC Revised Classification System: Stage 3-Defining Opportunistic Illnesses in HIV Infection

- Bacterial infections, multiple or recurrent*
- Candidiasis of bronchia, trachea, or lungs
- Candidiasis of esophagus
- Cervical cancer, invasive+
- Coccidioidomycosis, disseminated or extrapulmonary
- Cryptococcosis, extrapulmonary
- Cryptosporidiosis, chronic intestinal (>1 month)
- Cytomegalovirus disease (other than liver, spleen, or nodes), onset age > 1 month
- Cytomegalovirus retinitis (with loss of vision)
- Encephalopathy attributed to HIV^
- Herpes simplex: chronic ulcers (present for >1 month) or bronchitis, pneumonitis, or esophagitis (onset at age > 1 month)
- Histoplasmosis, disseminated or extrapulmonary
- Isosporiasis, chronic intestinal (> 1 month's duration)
- Kaposi's sarcoma
- Lymphoma, Burkitt's (or equivalent term)
- Lymphoma, immunoblastic (or equivalent term)
- Lymphoma, primary of brain
- *Mycobacterium avium* complex or *Mycobacterium kansasii*, disseminated or extrapulmonary
- *Mycobacterium tuberculosis* of any site, pulmonary+, disseminated, or extrapulmonary
- *Mycobacterium*, other species or unidentified species, disseminated or extrapulmonary
- *Pneumocystis jirovecii* (previously known as "*Pneumocystis carinii*") pneumonia
- Pneumonia, recurrent+
- Progressive multifocal leukoencephalopathy
- Salmonella septicemia, recurrent
- Toxoplasmosis of brain, onset at age > 1 month
- Wasting syndrome attributed to HIV

*Only among children aged < 6 years

+Only among adults, adolescents, and children aged ≥ 6 years

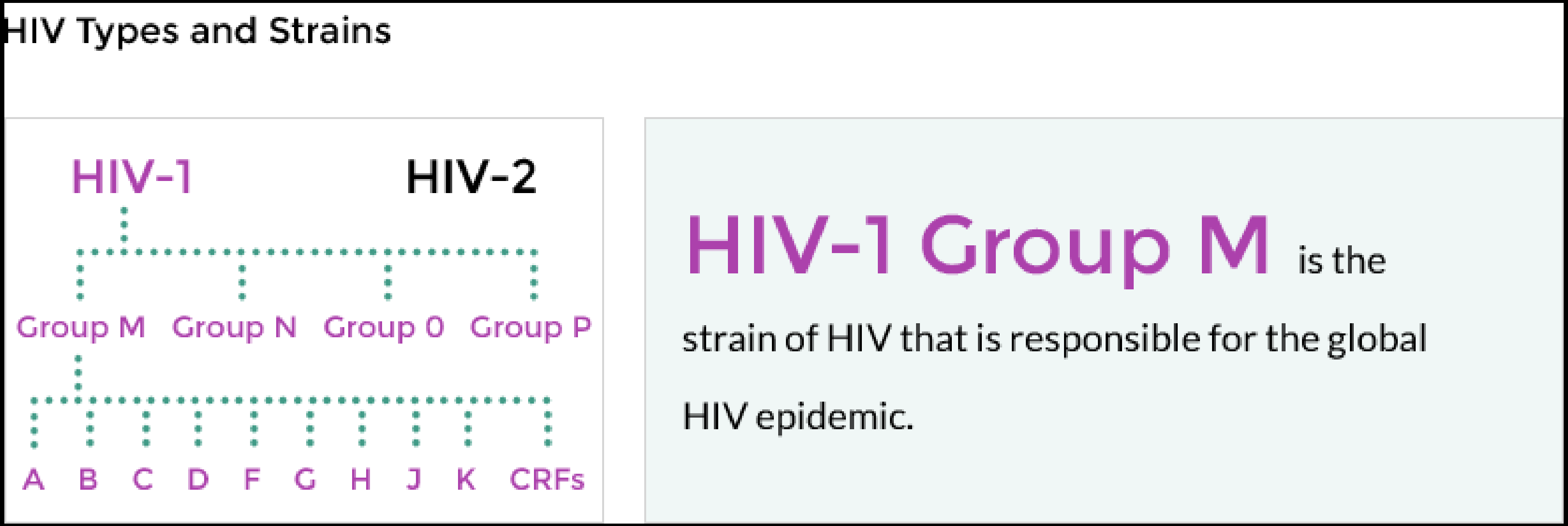
^Suggested diagnostic criteria for these illnesses are defined in prior surveillance case definitions

Causative Organism

Human Immunodeficiency Virus (**HIV*****(***

- Retrovirus (**2 copies of HIV RNA**(
- Characterized by
  - Profound Immunosuppression
  - Opportunistic infections, Secondary neoplasms
  - Neurological manifestations

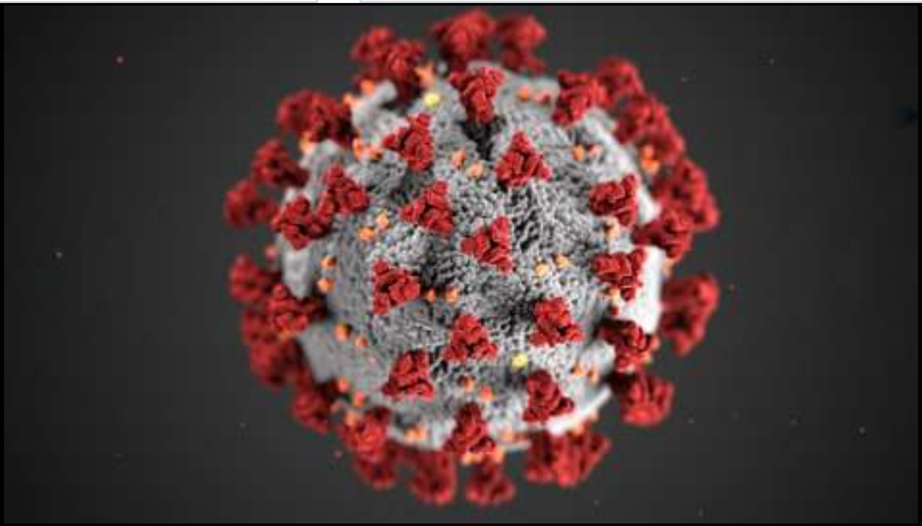
- There are two types of HIV:
    - **HIV-1**: World wide.
    - **HIV-2:** in West Africa & Sub-Saharan Africa .
- **HIV-2 is**
    - Less transmissible
    - Less pathogenic
    - May have a protective role against super infection with HIV-1



HIV Types and Strains

HIV-1
HIV-2
Group M
Group N
Group O
Group P
A
B
C
D
F
G
H
J
K
CRFs

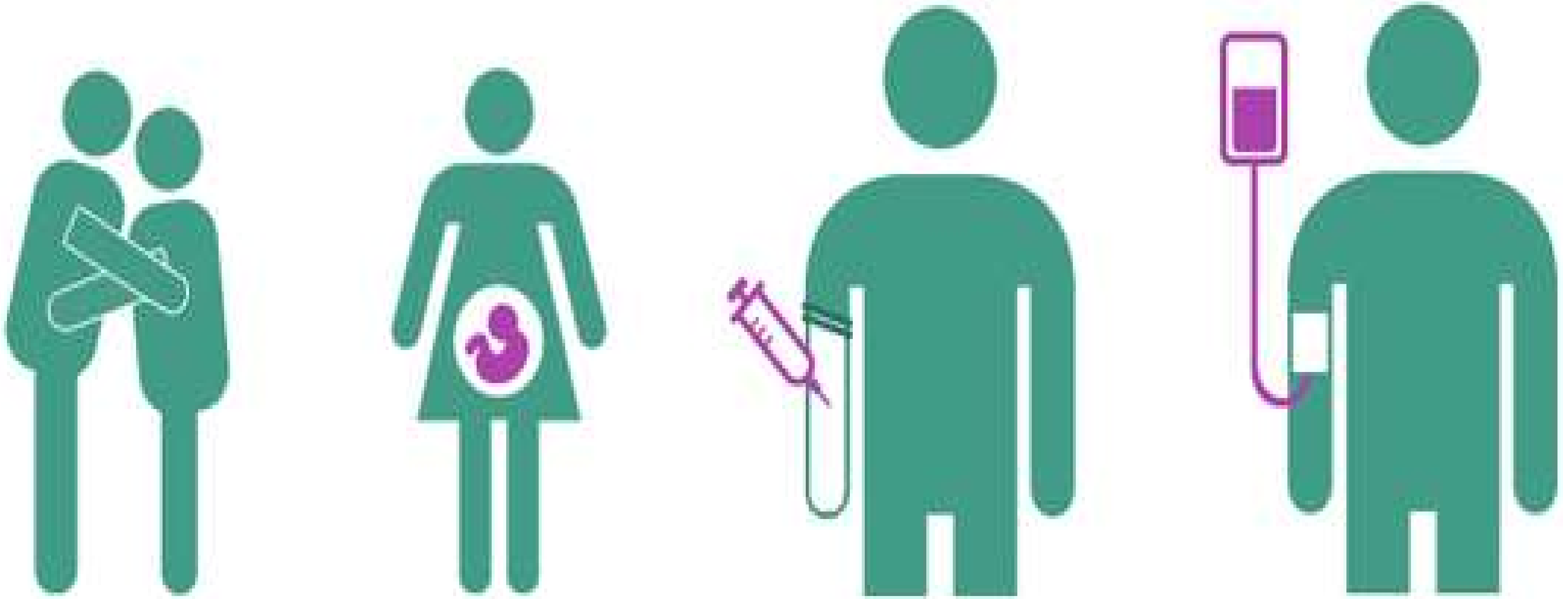
HIV-1 Group M is the strain of HIV that is responsible for the global HIV epidemic.



Mode of Transmission

- **Sexual Contact** ((75%
  - Homosexuals; MSM (Anal intercourse(
  - Heterosexuals (Vaginal intercourse(
  - Oral sex (extremely rare), Deep open-mouth kissing.
- **Infected mother to child** (**Vertical transmission**(
  - In utero
  - At birth
  - Breast milk
- **Parental Inoculation**:
  - Intravenous drug abusers
  - Recipients of blood and blood products (SCA, hemophiliacs). Dialysis…
  - Tattooing.
  - Accidental (health care workers(
- **Organ & Tissue donation**

YOU CAN GET HIV VIA...



Sex without
a condom

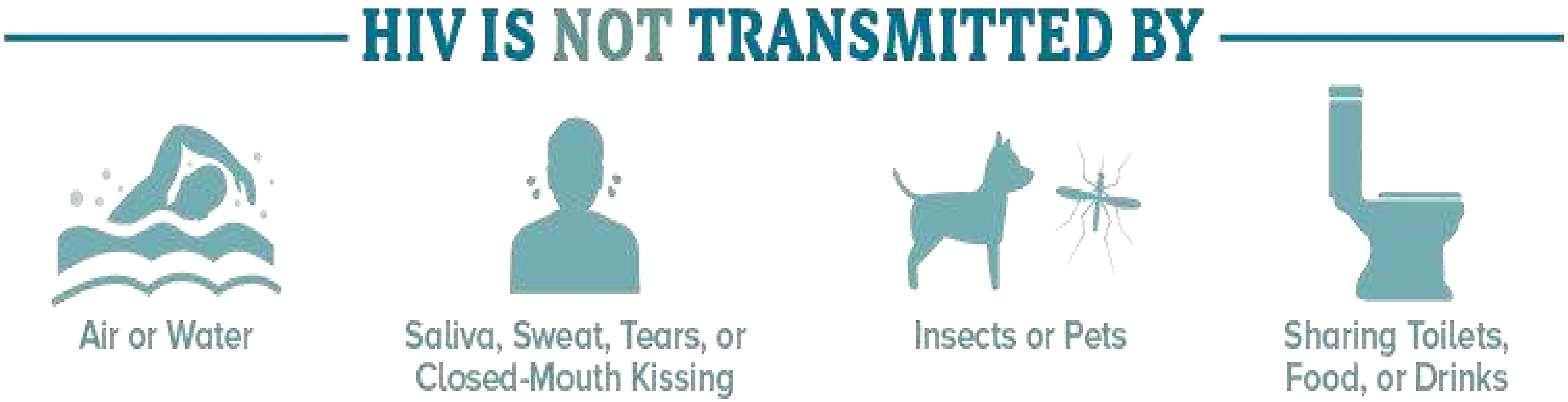
Passed from
mother to baby

Sharing injecting
equipment

Contaminated blood
transfusions &
organ transplants

Non proven modes of transmission

- Does not spread by any casual personal contact in the workplace, household or school (hugging, shaking hands, closed mouth kissing, toilet seats, food, water) or Air.


HIV IS NOT TRANSMITTED BY
Air or Water
Saliva, Sweat, Tears, or Closed-Mouth Kissing
Insects or Pets
Sharing Toilets, Food, or Drinks

- It is incompletely inactivated by treatment for 10 minutes at room temperature with any of the following : 50% ethanol , 35% isopropanol and 10% household bleach .It is also inactivated by extremes in PH or heating in liquid at 56^0C for 10 minutes.

- HIV can be transmitted through the following **body fluids**:
  - Blood
  - Semen and prosemen
  - Vaginal secretions
  - Rectal secretions
  - Breast milk

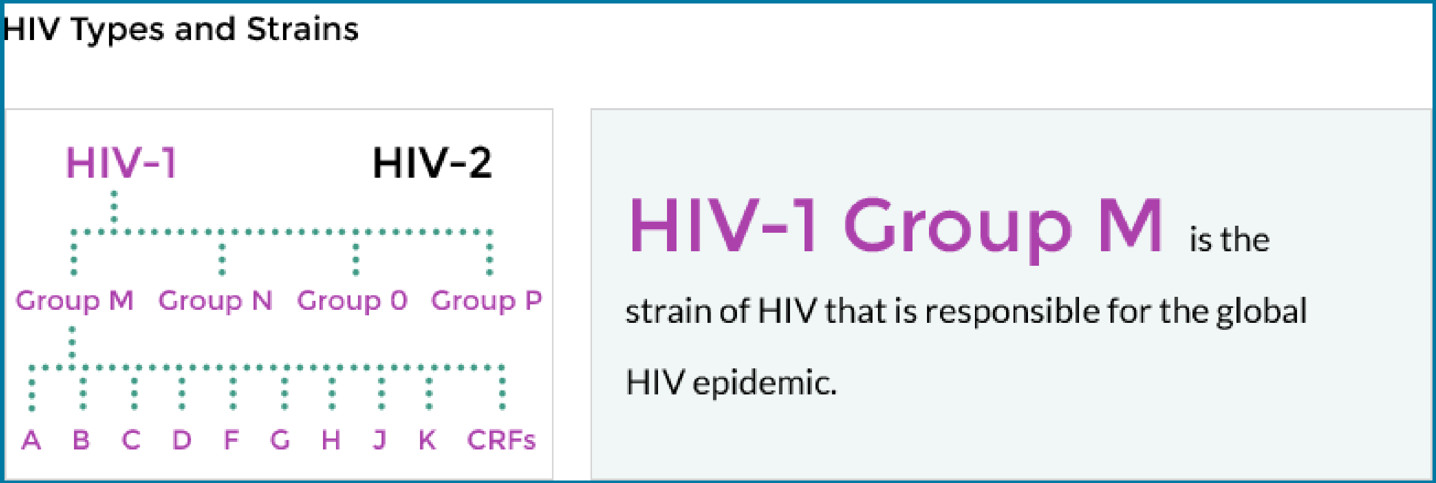
- These fluids must come in contact with:
  - Mucous membrane
  - Damaged tissue
  - Directly injected into the bloodstream.

- Mucous membranes are found inside the **rectum**, **vagina**, **penis**, and **mouth**.

- HIV can live in a used needle up to **42 days** depending on temperature and other factors

HIV Superinfection

- HIV superinfection is when a person with HIV gets infected with another strain of the virus. The new strain of HIV can replace the original strain or remain along with the original strain.


HIV Types and Strains

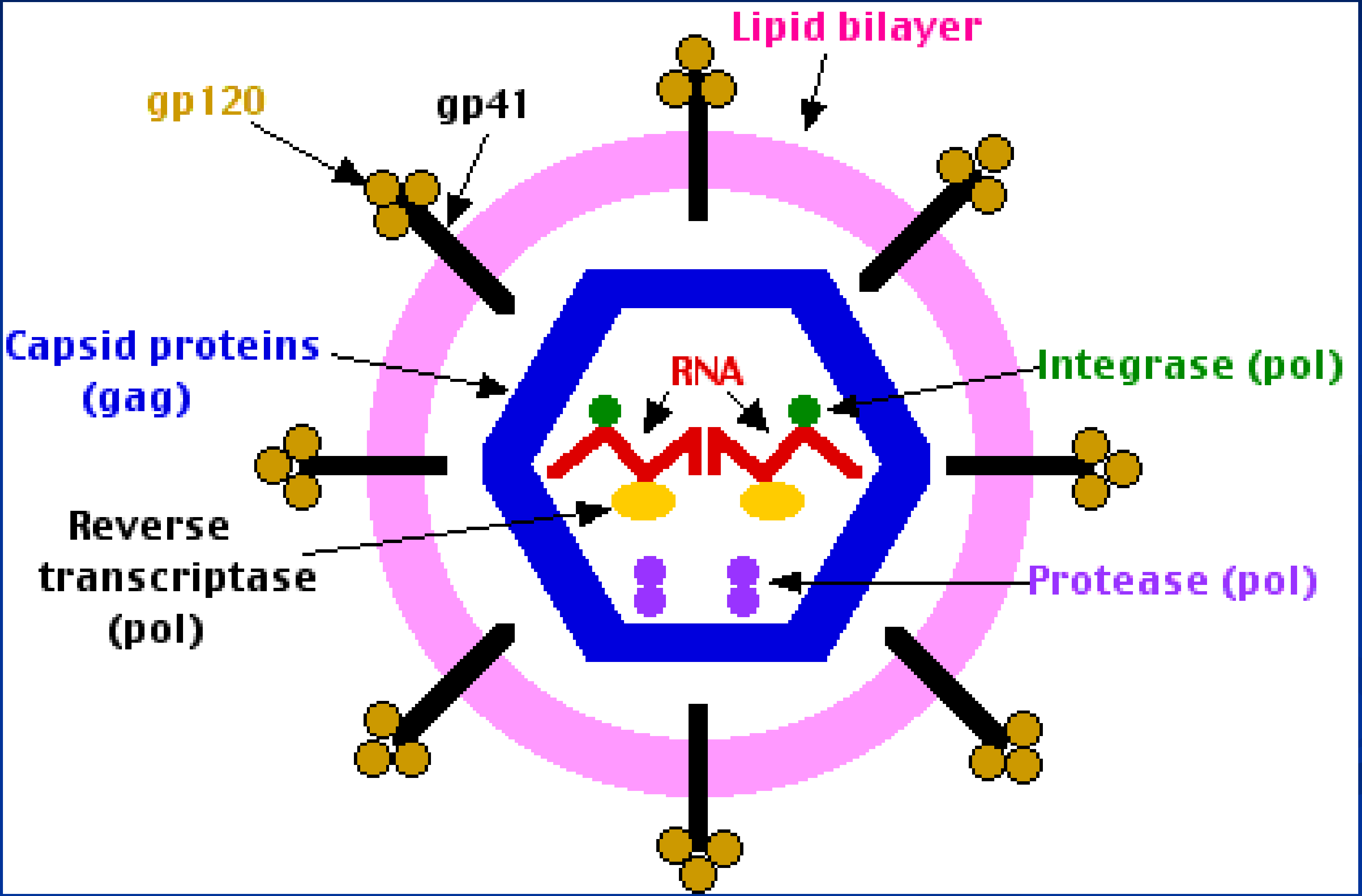
HIV-1
HIV-2
Group M
Group N
Group O
Group P
A
B
C
D
F
G
H
J
K
CRFs

HIV-1 Group M is the strain of HIV that is responsible for the global HIV epidemic.

HIV Structure

- Two copies of single-stranded RNA genome located inside a conical core which in turn is surrounded by a lipid envelope .
- Viral enzymes are:
  - **Reverse transcriptase** (changes RNA into DNA(
  - **Protease**
  - **Integrase** (integrates viral DNA into hosts DNA.(

HIV Structure

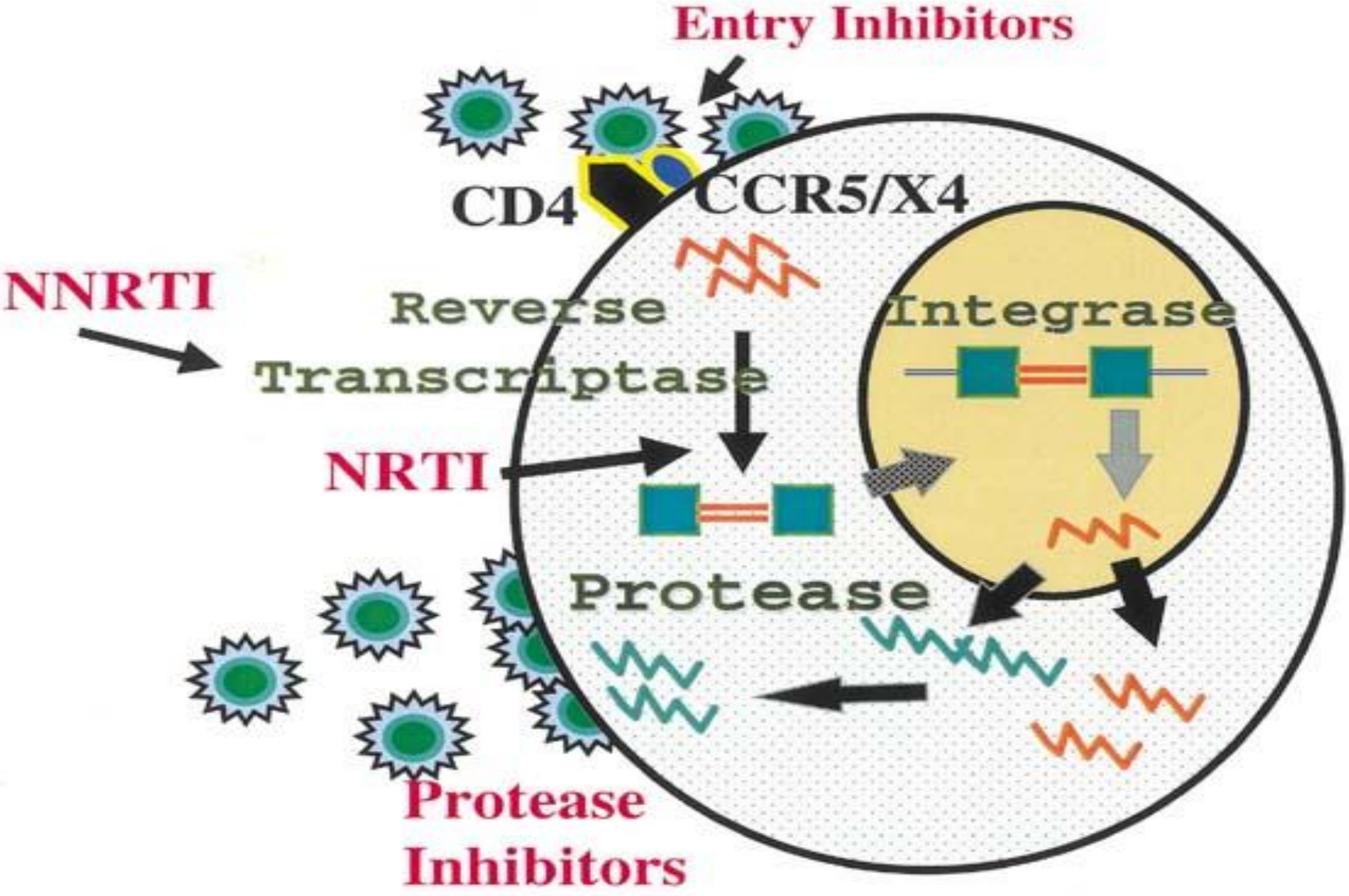

Lipid bilayer
gp120
gp41
Capsid proteins (gag)
RNA
Integrase (pol)
Reverse transcriptase (pol)
Protease (pol)

Life Cycle of HIV Virus & Pathogenesis

- Viral receptors:
  - CD4: helper T cells or macrophages, others (hemopoietic stem cells, endothelial cells(
  - Galactosyl ceramide: in nerve cells
- Life Cycle of HIV Virus & Pathogenesis:
  - **Binding** (to receptors & co-receptors) & **Entry** (fusion, endocytosis(
  - **Transcription**: into double stranded DNA by **Reverse Transcriptase**.
  - **Integration**: proviral DNA enters the nucleus and integrated into host DNA by **Integrase** enzyme.
  - **Viral replication & transcription**: within the host nucleus → many RNA genome & mRNA → transported to the cytoplasm
  - **Viral assembly**: formation of new viral particle by HIV **Protease**
  - **Viral budding**: infected cells release virions utilizing the host cell membrane to form virion outer envelop.

HIV leaves the host cell through:

-Budding
-Direct release after cell lysis
-Fusion to other non infected cells → multinucleated giant cells.



Entry Inhibitors

CD4

CCR5/X4

NNRTI

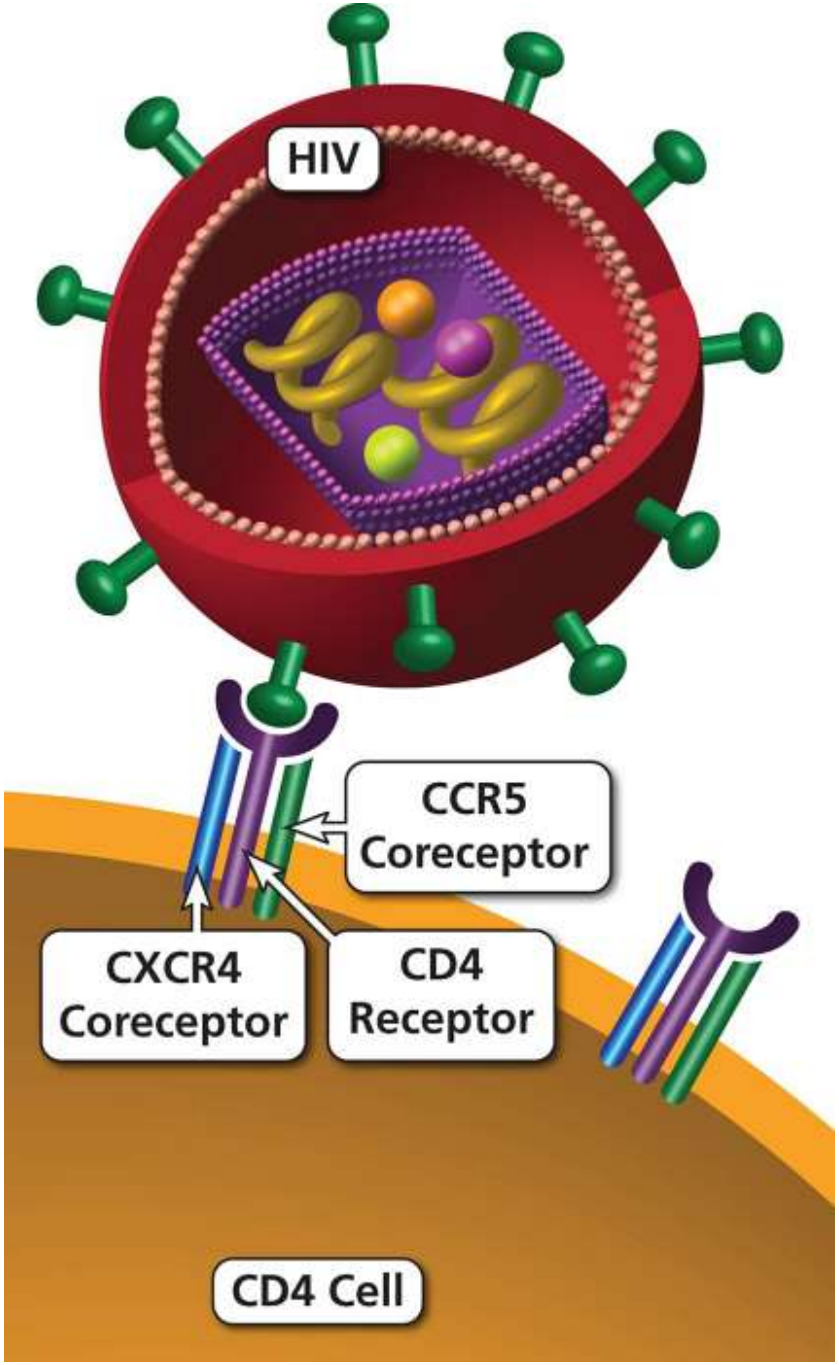
Reverse Transcriptase

Integrase

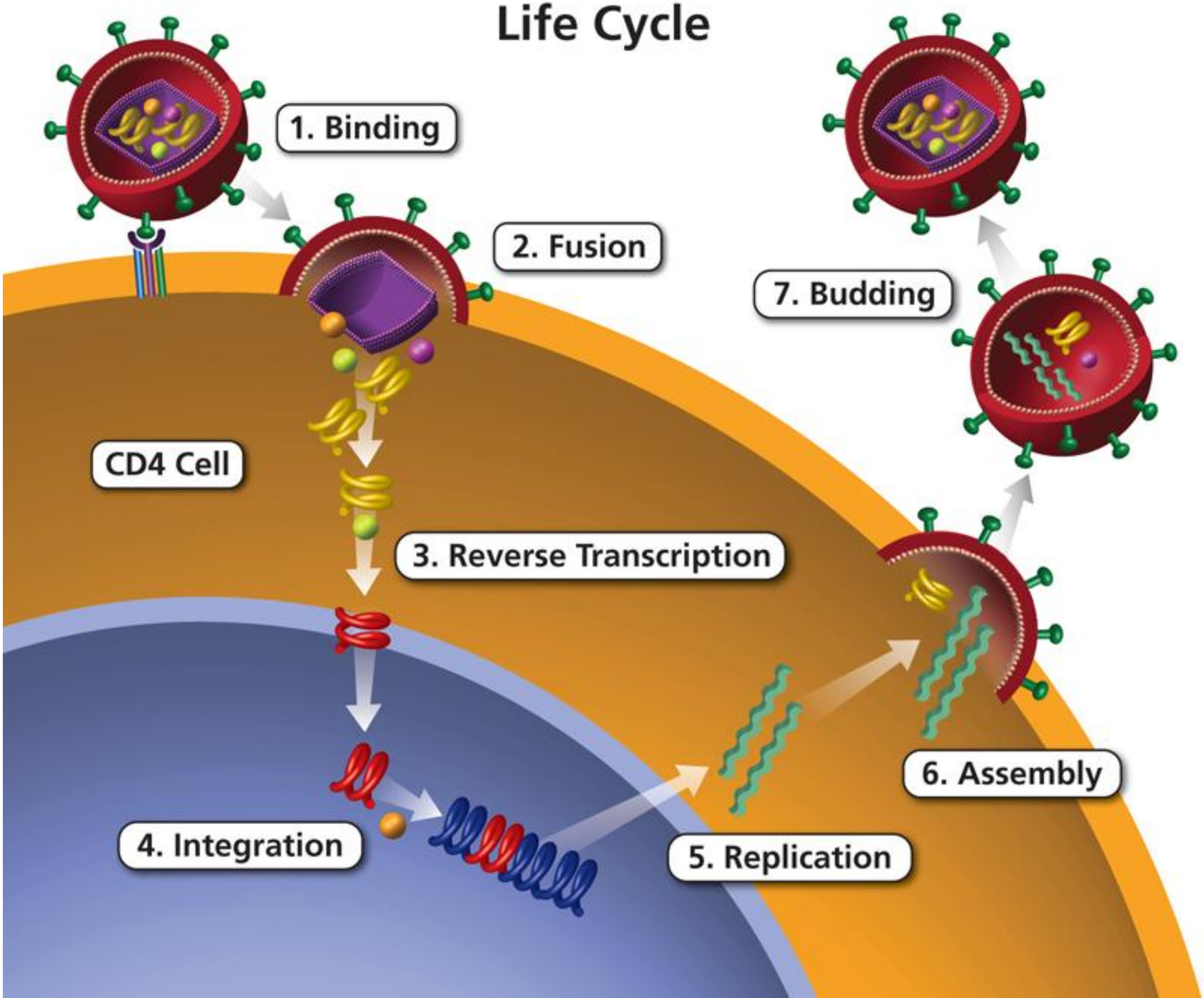
NRTI

Protease

Protease Inhibitors


HIV
CCR5 Coreceptor
CXCR4 Coreceptor
CD4 Receptor
CD4 Cell

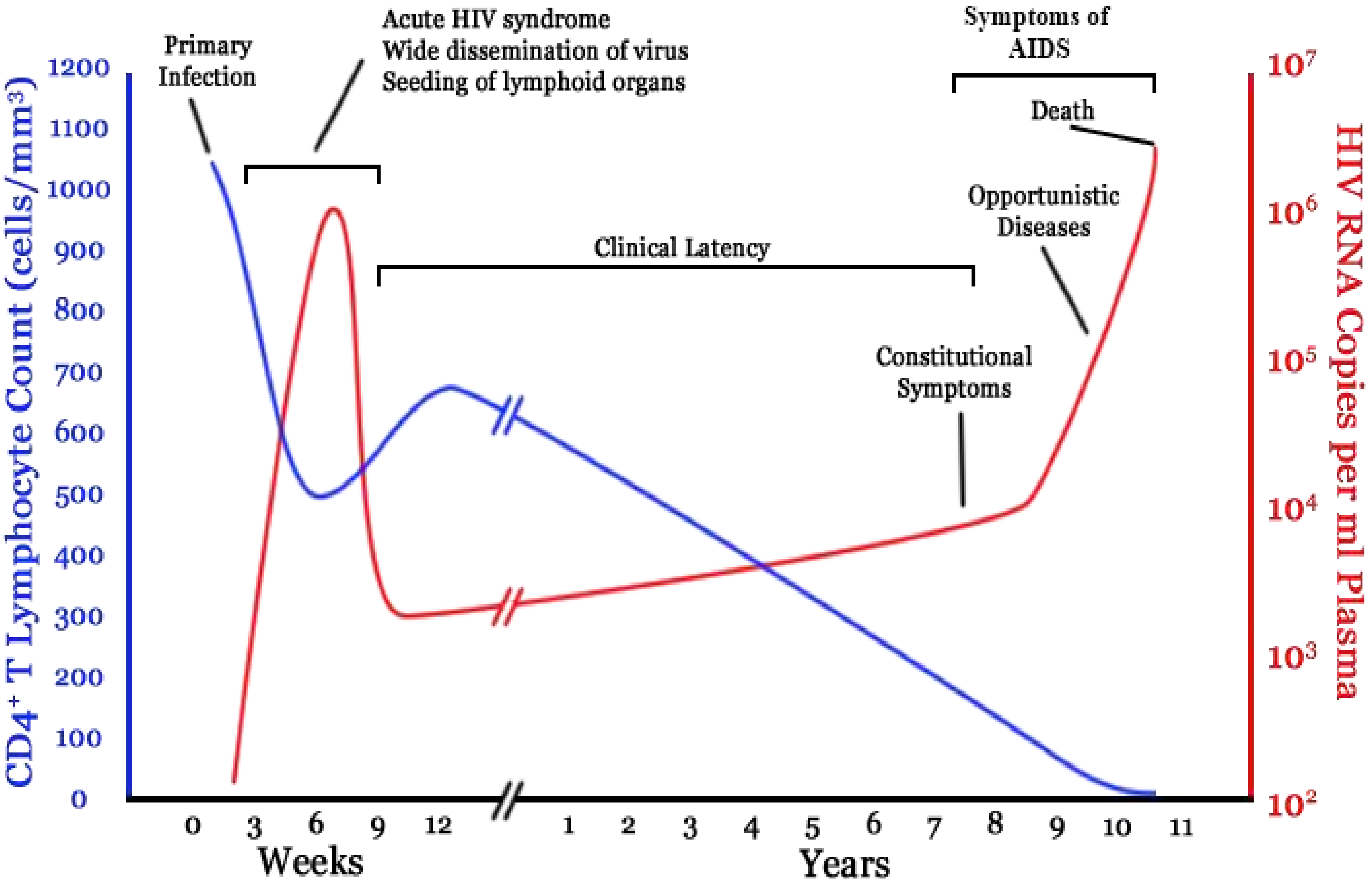
Life Cycle


1. Binding
2. Fusion
CD4 Cell
3. Reverse Transcription
4. Integration
5. Replication
6. Assembly
7. Budding

Natural History of HIV

- Primary (Acute) HIV infection = *The time period from initial infection with HIV to the development of an antibody response (**seroconversion**).* **Most infectious stage**.
- Asymptomatic HIV infection
- Early symptomatic HIV infection
- Late symptomatic HIV infection
- Advanced HIV infection (AIDS(

Natural History of HIV



Primary HIV infection

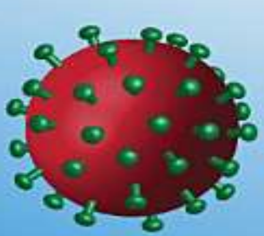
Clinical picture (2-4 weeks :(

- Symptomatic in 50-90%
- Acute onset of fever, generalized L.N. enlargement, pharyngitis, arthralgia, myalgia, headache, malaise, diarrhea, maculopapular rash.
- Oral and genital ulceration.
- Severe neurological manifestations e.g.aseptic meningitis, encephalitis
- Opportunistic infections e.g. oral & esophageal candida

Laboratory findings:

- ↓ CD4 count (< 200 cells/mm (^{3}

HIV and AIDS:
What's the difference?

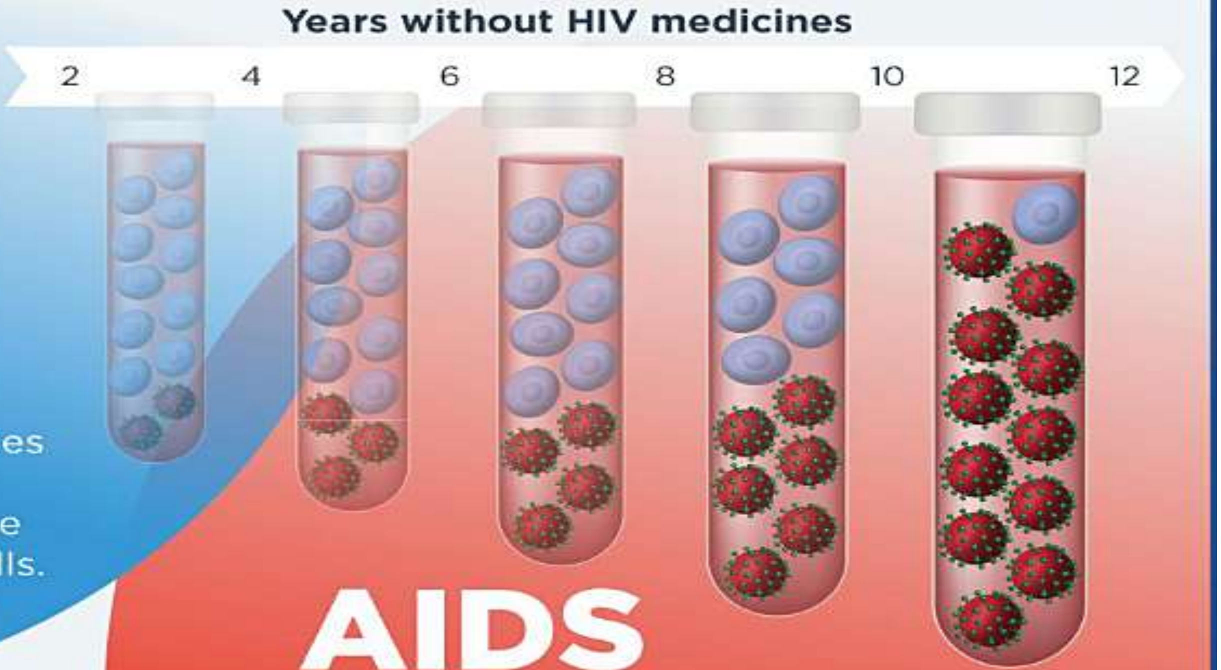


HIV

- HIV is the virus that causes HIV infection.
- HIV damages the immune system by killing CD4 cells.

CD4 Cells

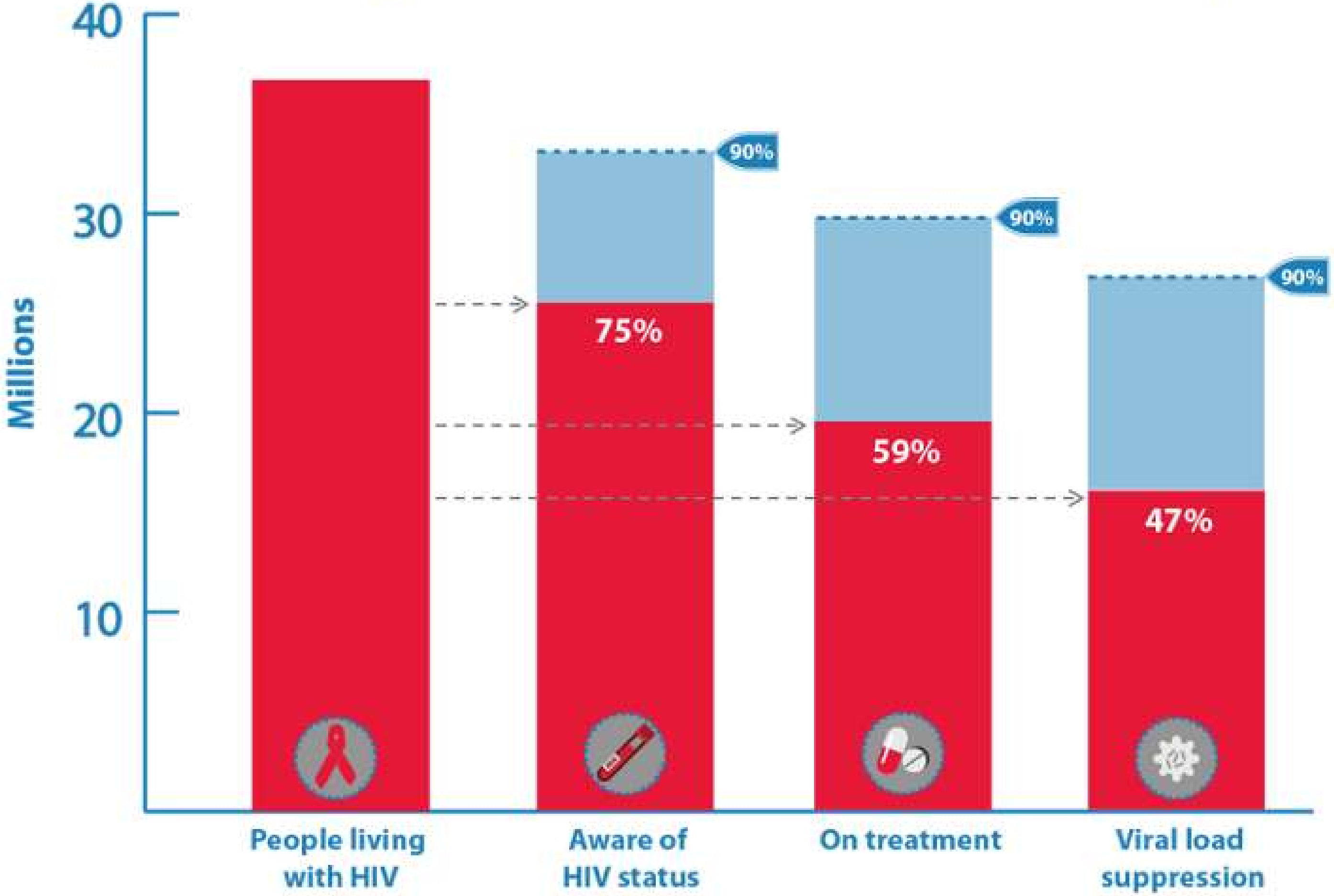
- CD4 cells are part of the immune system.
- HIV attacks and kills CD4 cells.
- Loss of CD4 cells makes it hard for the body to fight off infections.



AIDS

- AIDS is the last stage of HIV infection.
- As HIV infection advances to AIDS, the amount of HIV in the body increases and the number of CD4 cells decreases.
- HIV medicines can stop HIV infection from progressing to AIDS.
- Without HIV medicines, HIV advances to AIDS in about 10 to 12 years.

HIV testing and care continuum (2017)



Source: UNAIDS/WHO estimates

World Health Organization

Asymptomatic HIV infection

Clinical picture (2-15 years:(

- No HIV related symptoms
- Generalized L.N. enlargement
- Skin manifestations
- Seborrheic dermatitis

Lab. Finding :

- CD4 count >500 cells / mm.^{3}
- Rate of drop 40 – 80 cells / mm^3 per year .

Early symptomatic HIV infection

Clinical picture:

- Constitutional manif.: fever, diarrhea, weight loss, headache, fatigue.
- Cutaneous manifestations: candidiasis (oral & vulvovaginal), herpes zoster, herpes simplex, oral hairy leukoplakia.
- Generalized L.N. enlargement
- Opportunistic infection: sinusitis, bronchitis, pneumonia
- Idiopathic thrombocytopenic purpura.- Kaposi sarcoma .

Lab:

- CD4 200 – 500 cells / $mm.^3$

Late symptomatic HIV infection

Clinical picture ***:***

- Opportunistic infection e.g. Pneumocystits carnii pneumonia, toxoplasmosis...elc.
- Neoplastic e.g. Lymphoma, Kaposi sarcoma
- Constitutional symptoms (AIDS wasting(
- Neurological e.g.Polyneuropathy, AIDS dementia complex (ADC . (

Lab:

- CD4 50 – 200 cells / mm.^{3}

Advanced HIV infection

Clinical picture:

- **All AIDS defining Opportunistic infection and malignancies.**

Lab:

- CD4 < 50 cells / mm^3

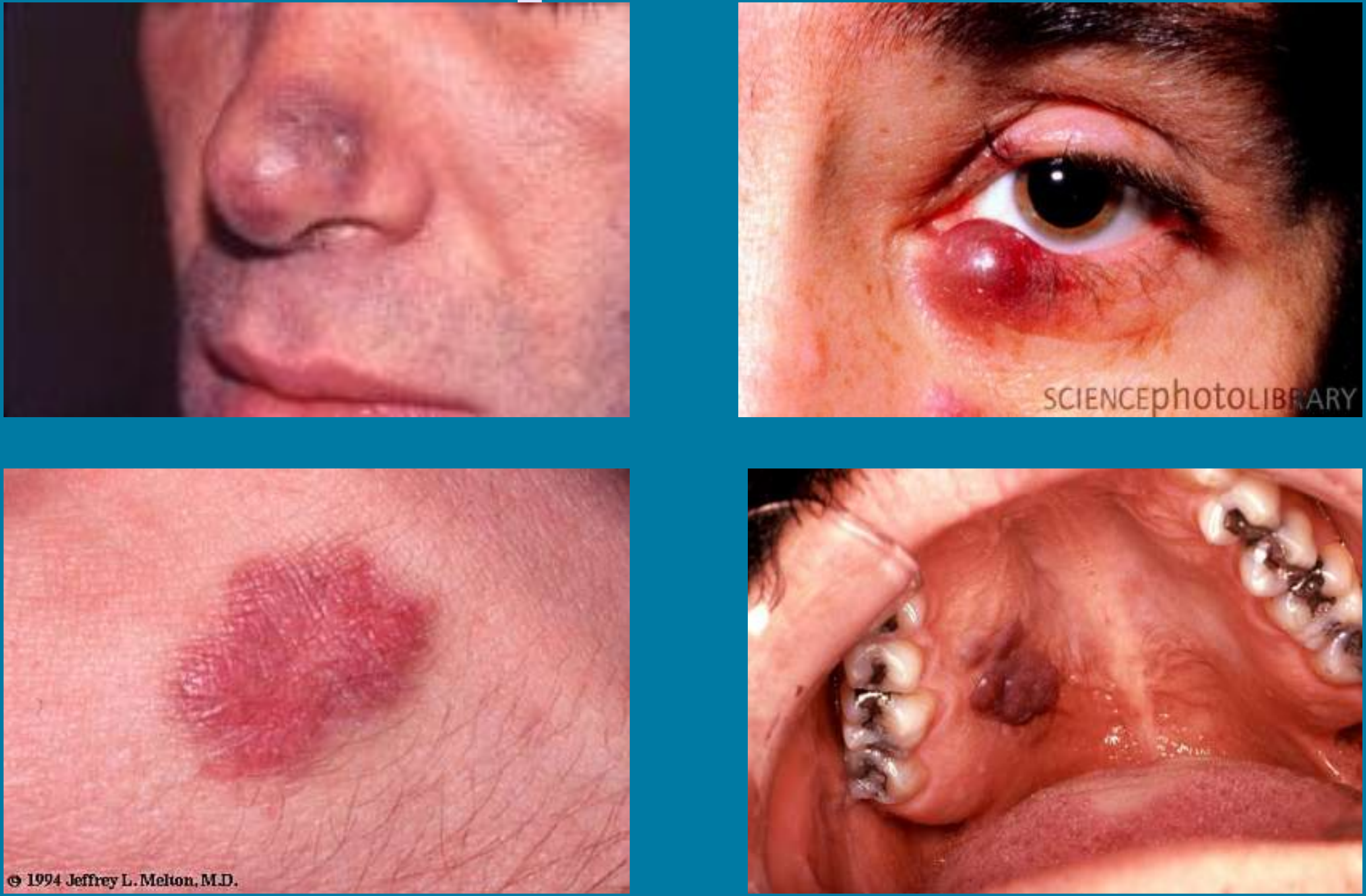
Manifestation of AIDS

- **Oral manifestations:**
  - *Oral candidiasis*: appears as cheesy white membrane on the mouth cavity and angles of the mouth. This is a strong predictor for both progression of the disease and its mortality irrespective of the laboratory CD4 cell count. Pharyngitis or oesophygeal candidiasis may occur (dysphagia, hurt burn, dyspepsia.(
  - *Oral hairy leukoplakia*: caused by Epstein-Barr virus. Appears as grayish-white corrugated thick lesions on the sides of the tongue that cannot be scrapped (in contrast to the oral thrush.(
  - *Recurrent HSV infections.*

Dermatological manifestations

-**Non malignant**	-**Malignant conditions**
(Infectious & non infectious) -**Seborrhoeic dermatitis** (**The most common dermatological manifestation**) -Folliculitis (intensely pruritic) -Viral, bacterial, fungal	-**Kaposi sarcoma** (HHV8, **commonest Malignancy**(-BCC -SCC -Melanoma

Kaposi Sarcoma



SCIENCEphotoLIBRARY

© 1994 Jeffrey L. Melton, M.D.

Neurological manifestations

- AIDS dementia complex (ADC:(
  - The commonest neurological manifestation (20% of AIDS patients.(
  - There is cognitive deterioration, declining motor performance, behavioral changes, and convulsions. Death occurs within 6 months .
- HIV encephalopathy
- Myelopathy
- CMV Encephalitis , Retinitis: CMV is the commonest cause of blindness in AIDS
- Meningitis
- Space occupying lesions (cerebral toxoplasmosis, Primary cerebral lymphoma(
- Psychiatric disease

Respiratory manifestations

- **Pulmonary Infections**:
  - **Pneumocystis carinii pneumonia** (PCP) is the most common opportunistic infection and is the presenting feature in 50% of all cases of AIDS
  - TB
  - Lymphocytic interstitial pneumonitis (in children).
- **Pulmonary Malignancies:**
  - Kaposi's sarcoma, Lung carcinoma

Gastrointestinal manifestations

- Opportunistic and malignant diseases in AIDS patients may affect any part of the GIT. The most common symptoms include oral ulcerations, dysphagia, and weight loss that may be caused by Candida affecting most parts of the GIT, herpes virus, or Epstein-Barr virus.
- In Africa AIDS the main presentation is the slim disease with sever uncontrolled diarrhea as the main symptom in over 70% of the cases .

Renal Manifestations

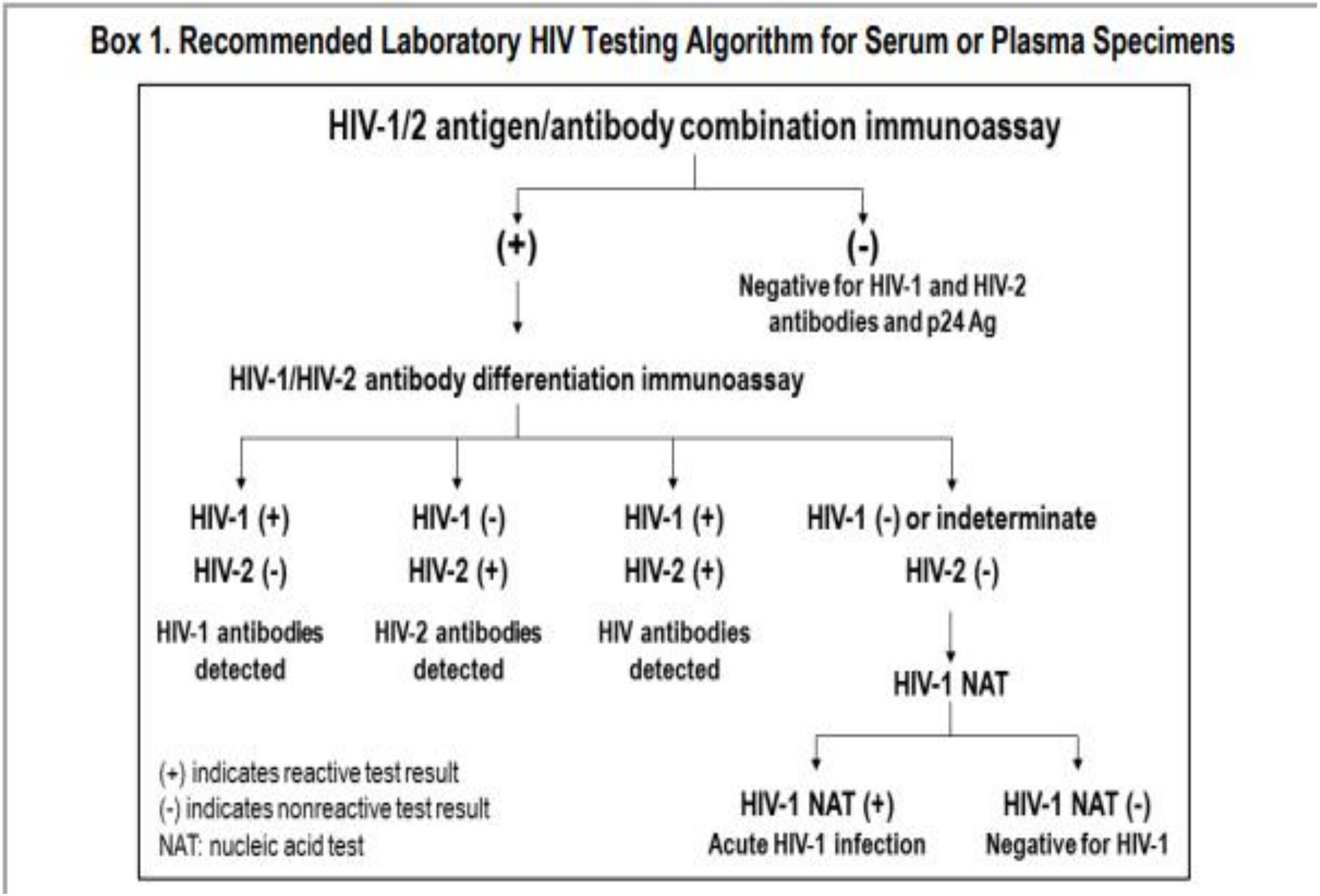
- *Prerenal*: Volume depletion (hypovolemic shock) and electrolyte imbalance (due to hemorrhage, diarrhea, vomiting) →Acute tubular necrosis.
- *Renal*: 1ry HIV nephropathy →Nephrotic syndrome (proteinuria & oedema) →CRF .

Male Genital Tract

- About half of male AIDS patients have clinical evidence of gonadal dysfunction with decreased libido and impotence.
- **Erectile dysfunction** :
  - Psychogenic: fatigue, depression→libido
  - Neurogenic: viral myelitis, malig.
  - Hormonal: hypogonadism (↓Testo(.
- **Infertility (Testicular atrophy:(**
  - Chronic illness, fever, malnutrition
  - Abnormal HPG axis
  - HIV itself → cytopathic effect on germ cells.
  - 2ry causes e.g., CMV
  - Medications: long term antibiotics, antifungals, chemotherapy.

Investigation

- **Virus detection**
  - Antigen/antibody combination immunoassay
  - Antibody immunoassay
  - HIV-1 nucleic acid test (NAT(
- **CD4 cell count** & CD4/CD8 ratio
- CBC: Pancytopenia
- Others



Box 1. Recommended Laboratory HIV Testing Algorithm for Serum or Plasma Specimens

HIV-1/2 antigen/antibody combination immunoassay

(+)

(-)
Negative for HIV-1 and HIV-2 antibodies and p24 Ag

HIV-1/HIV-2 antibody differentiation immunoassay

HIV-1 (+)
HIV-2 (-)
HIV-1 antibodies detected

HIV-1 (-)
HIV-2 (+)
HIV-2 antibodies detected

HIV-1 (+)
HIV-2 (+)
HIV antibodies detected

HIV-1 (-) or indeterminate
HIV-2 (-)

HIV-1 NAT

HIV-1 NAT (+)
Acute HIV-1 infection

HIV-1 NAT (-)
Negative for HIV-1

(+) indicates reactive test result
(-) indicates nonreactive test result
NAT: nucleic acid test

- **No HIV test can detect HIV immediately after infection.**
- The time between when a person may have been exposed to HIV and when a test can tell for sure whether they have HIV is called the ***window period***. The window period varies from person to person and depends on the type of test used to detect HIV

- A **nucleic acid test (NAT)** can usually tell you if you have HIV infection 10 to 33 days after an exposure.
- An **antigen/antibody test** performed by a laboratory on blood from a vein can usually detect HIV infection 18 to 45 days after an exposure.

- Antigen/ antibody tests done with blood from a finger prick can take longer to detect HIV (18 to 90 days after an exposure.(
- When the goal is to tell for sure that a person does not have HIV, an antigen/antibody test performed by a laboratory on blood from a vein is preferred.

Treatment

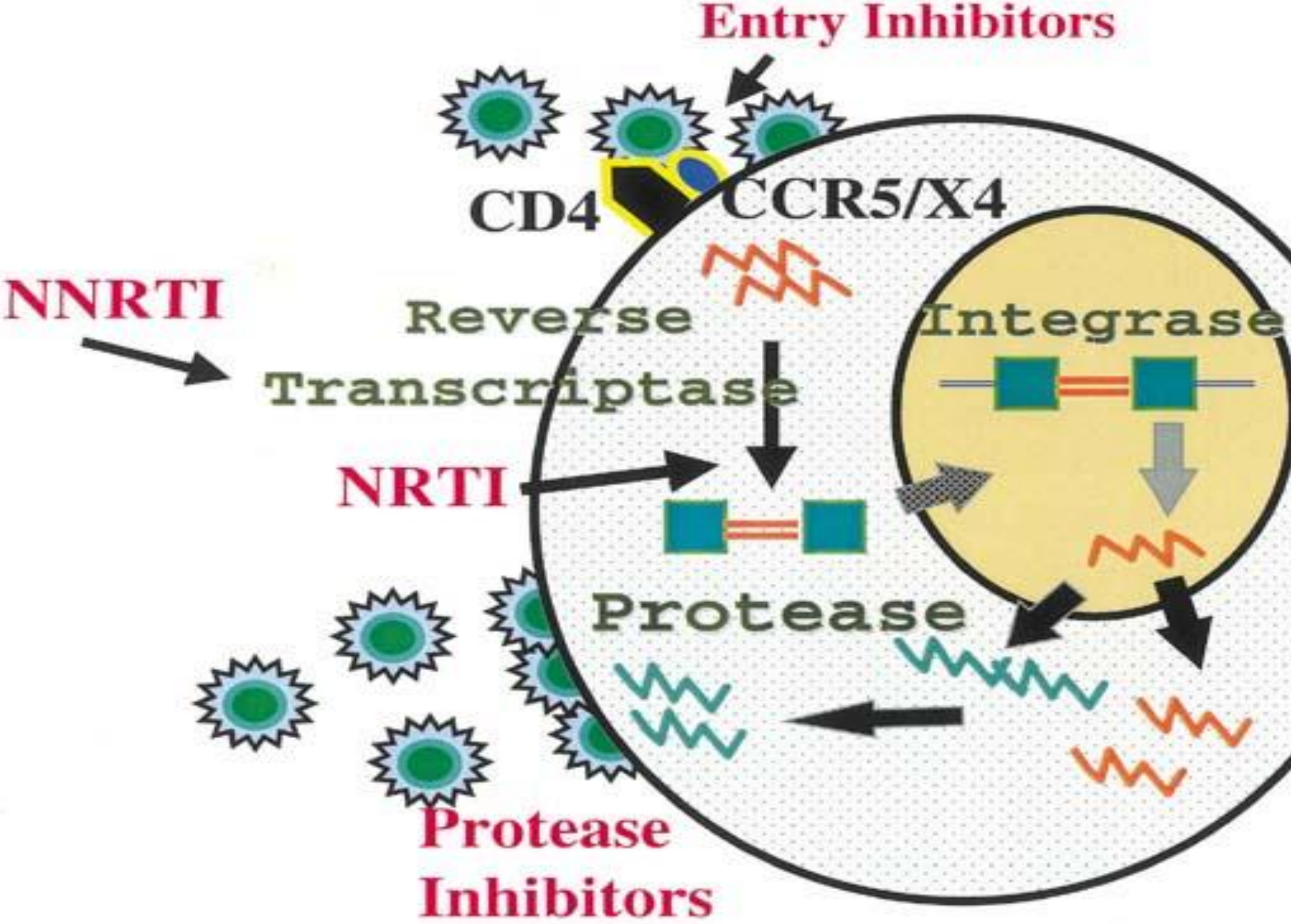
ART should be initiated **as soon as** possible for all persons with HIV infection regardless of CD4+ T-cell count, both for **individual health** and to **prevent HIV transmission.**

Persons with HIV infection who achieve and maintain a viral load suppressed to <200 copies/mL with ART have effectively no risk for sexually transmitting HIV

NAT: It resulted in a significant reduction of the HIV-1 window period, from 22 days without NAT to 9–12 days with NAT

As of 2020, the FDA has approved 222 Antiretroviral Drugs for Global HIV/AIDS Relief .

The human body **cannot get rid** of the virus and **no effective cure exists**. However, with proper medical care, HIV can be controlled with HIV medicines.


Entry Inhibitors
CD4
CCR5/X4
NNRTI
Reverse Transcriptase
Integrase
NRTI
Protease
Protease Inhibitors

Treatment

- 1Prevention is the only 100% therapy for HIV

- Prevention of sexual transmission: Sex education (Refrain from premarital & extramarital sexual relations, safe sex…how(؟
- Prevention of non-sexual transmission:
  - Strict examination of blood during donation
  - Reduce risk of occupational exposure to body fluids e.g by using gloves, gowns, masks, protective eye wear
- Prevention of vertical transmission (before, during & after delivery.(
- Vaccines (under trial(

Treatment

When to start treatment؟

- ***Adults*** (viral load 5000-10,000 copies/ml, CD4 <200,and clinical signs(
- ***Pregnant*** (14th week of gestation or soon after that) + contraception other than IUD.
- ***Newborn*** of infected mother (at birth until 6 weeks(
- ***Partner (s)*** of infected persons
- ***Post-exposure prophylaxis; PEP*** (as soon as possible: within 72 hrs for 28 days(

Treatment

*- 2*Antiretroviral Therapy (ART:(

These drugs inhibits the different viral enzymes leading to reduction of viral replication but not total eradication of HIV.

They include:

- Entry & Fusion Inhibitors: e.g., Fuzeon.
- Nucleoside/Nucleotide reverse transcriptase inhibitors (NRTI/NtRTI): e.g., Zidovudine
- Non Nucleoside/Nucleotide Reverse Transcriptase Inhibitors)NNRTI): e.g., Nevirapine
- Protease Inhibitors (PI): e.g., Saquinavir
- Integrase Inhibitors (under trial.(

--The antiretroviral therapy consists of a combination of two or more drugs to increase the drug effect (Highly active antiretroviral therapy; HAART(

--Drug resistance is a major problem due to the high mutation capability of HIV .

PrEP

A course of ART to HIV negative people.

PrEP is recommended for populations who are at higher risk of HIV .

- Men who have sex with men (MSM(
- Sex workers
- People who use drugs
- Young women in southern Africa.

Elite controllers; those who can live without treatment depending on their immunity

Treatment

-3**Antibodies against HIV (oct, (2016**

-4Immunization: PCP, HBV, Influenza, DPT… NB: live attenuated vaccines are contraindicated, except MMR for children.

-5***Prevention and treatment of opportunistic infections.***

-6***Treatment of associated morbidity (weight loss, malignancy…etc.)….Nutritional support.***

Thank You

Nucleic acid-based tests to detect HIV infection include DNA PCR tests targeted to detect integrated proviral DNA and RNA RT-PCR tests that detect viral RNA. RNA RT-PCR tests may also have the potential to detect integrated proviral DNA

Elite controllers ; those who can live longer lives without treatment depending on their immunity.

الإيدز ليس فقط عقاب الله للمثليين إنه عقاب الله على المجتمع الذي يتسامح مع مثليون جنسيا.

جيري فالويل

As Long as
it's about **HIV**,
hate the disease, but not
the diseased.
Spread awareness, not
ignorance.

©WishesMsg.com

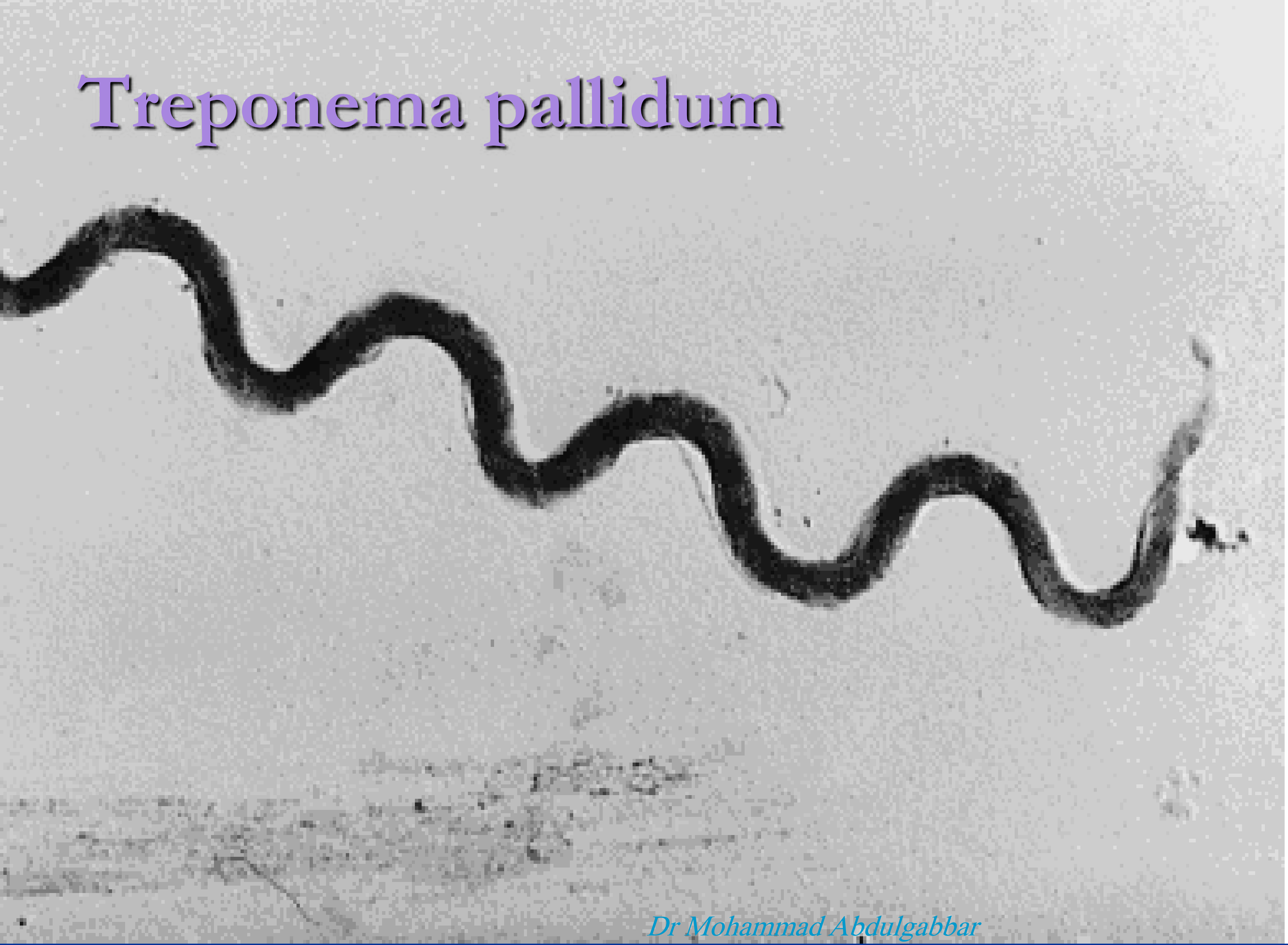
SYPHILIS

Causative Organism

Treponema pallidum (by Dark ground microscope):

- Spirochaete
- Spiral organism with regular coils.
- Moves in a "cork-screw" fashion.
- Multiply by transverse fission every 30 hours.
- Cannot be grown on ordinary culture media and needs animal inoculation (rabbits, hamsters).
- Susceptible to dryness, heat, weak antiseptics.

Treponema pallidum



Modes of transmission

1. Sexual contact: (needs hotness, humidity, simple abrasion). Hetero & Homosexuals.
2. Asexual contact:
   - *Accidental*: Physicians, Nurses, Lab workers, Bl.transfusion ⇨ syphilis demblee
   - *Vertical*: Trans-placental, vaginal delivery, Breast feeding

Pathology

Early (organism enters the body through abrasion in skin or MM):

- Cellular infiltration: **Plasma** cells, lymphocytes (dermis) ⇨ dull red **papule**

- Vascular reaction: Endarteritis obliterans (Intima ⇨ thick, hypertrophic) thrombotic obstruction of blood vessels ⇨ **Ulcer** (necrosis of overlying epidermis).

- Organism ⇨ LN ⇨ Blood stream

Late:

- **Balanced State: If disturbed** ⇨ GUMMA (Central area of necrosis (DD= TB), surrounded by granulation tissue, surrounded by narrow zone of fibrous tissue).

⇨ Vascular affection

⇨ Neurological affection

Classification of Syphilis

I – **Acquired Syphilis**:

1. **Early** (< 2years, Infective)
   - Primary ⇨ Chancre
   - Secondary ⇨ Generalized affection.
   - Early latent ⇨ Asymptomatic
2. **Late** (> 2years, Not infective)
   - Late latent ⇨ Asymptomatic
   - Benign tertiary ⇨ GUMMA
   - Malignant tertiary
     - Cardiovascular $
     - Neuro $

II – **Congenital Syphilis**:

1. **Early** (< 2years, Infective)
   - No Primary
   - =Secondary
2. **Late** (> 2years, Not infective)
   - =tertiary
   - Neuro $ & V.Rare Cardiovascular $
3. Stigmata: permanent scars

Dr Mohammad Abdulgabbar

Acquired Syphilis

Primary $:

- IP: 9-90 days.
- Dull red painless PAPULE ⇨ Painless ULCER.
- CHANCRE:
  - Site: Genital (90%) ♀ ♂ & Extragenital
  - Character
- Regional LN

D.D.: Genital ulcer

Primary Syphilis

Inspection

Chancre

Palpation

LNs

DD

??

Chancre redux

$ démblé

Bubon démblé

Pseudochancre redux

Chancre

Inspection

Number: Single

Sites:(Genital, Extragenital)

Size: (0.5-2cm in Diam'.)

Shape: rounded or ovale

Edge: raised at periphery & slopping towards center)

Floor: clean,pale,red & oozing serous fluid.

Palpation

Base: Indurated

Tenderness: Absent ??

Fixity: Not fixed to deep structures.

FATE

Slow healing without ttt in 3-8wks with a thin scar.

LNs: Bil, Sym, Not tender,Rubbery, Mobile, Overlying skin:Normal

DD of Genital Ulcer

STDs

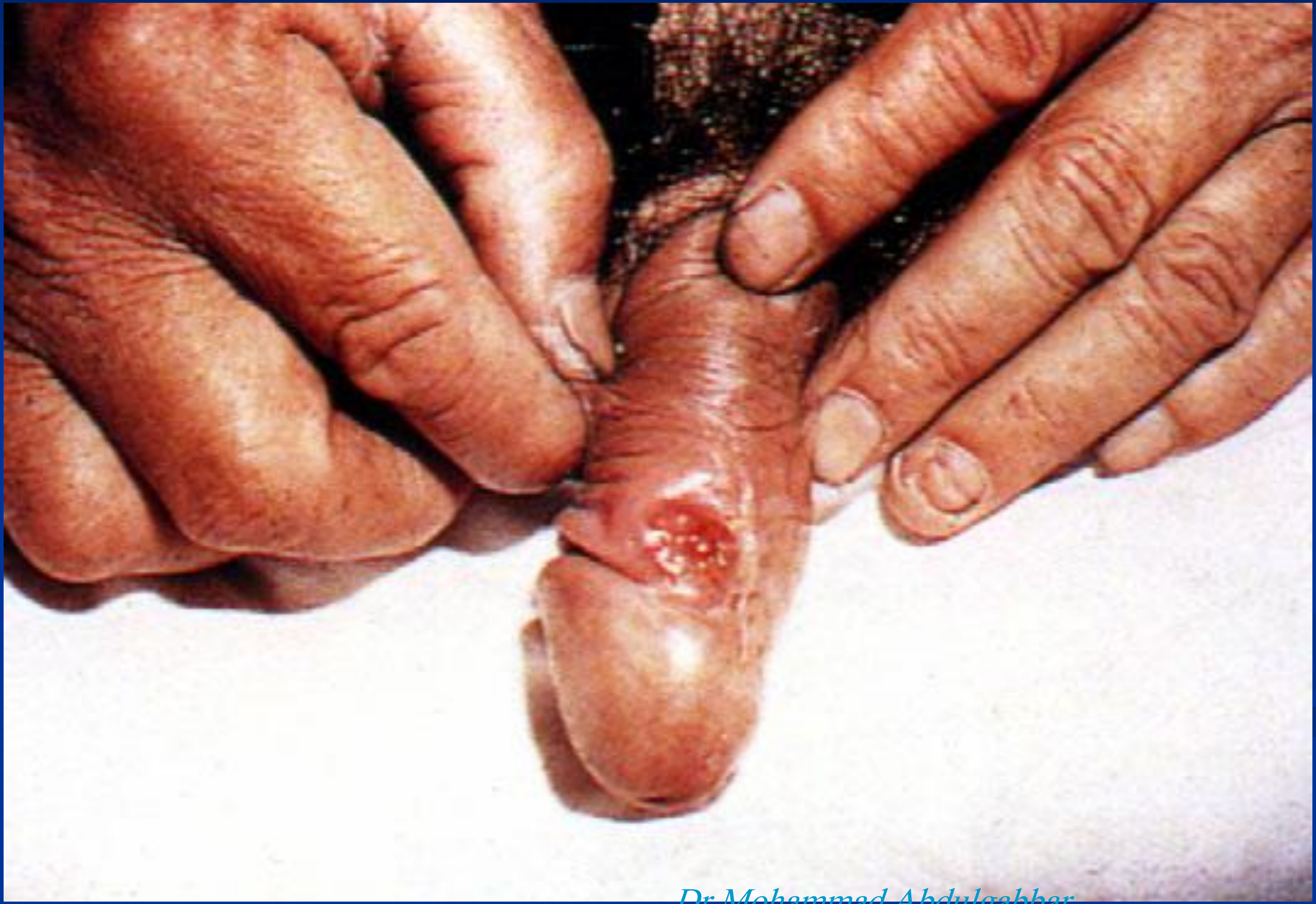
- Syphilis: 1) Chancre
  - 2) Mucous patch ulceration in 2ry $
  - 3) Gummatous ulcers in tertiary $
- Chancroid (H. Ducreyi)
- Lymphogranuloma venerum (Chlamydia T.)
- Granuloma inguinale (Calymmatobacterium granulomatis)
- Herpes progenitalis (HSV)
- Primary HIV ulcers (non-specific)

Non-STDs

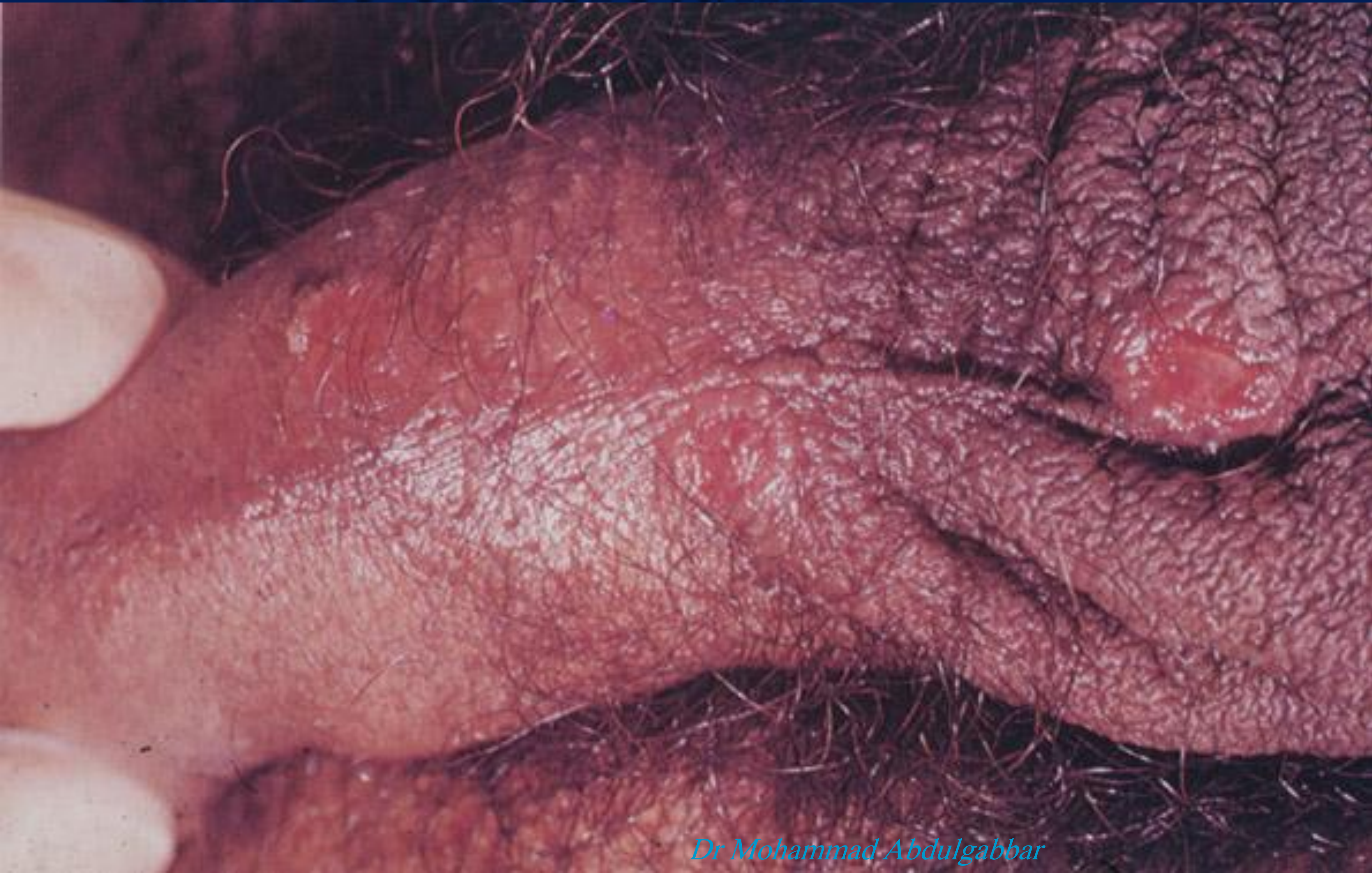
- Traumatic
- Infective (TB, Bilh, H.zoster…)
- Malignant
- Behcet syndrome
- Fixed drug eruption

Dr Mohammad Abdulgabbar

Chancre on penile shaft



Chancre on scrotum



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Diagnosis of 1ry Syphilis

- **Darkground Microscopy**: the only sure method
  -Idea
  -Material: Ulcer, LN
  -If –ve repeat daily for three days.
  -Advantages / Disadvantages

- **Serological** Tests: +ve in 50%, if –ve repeat/wk for 3 wks then /month for 3 months

Secondary $:

- ~7-10 wks after infection or 6-8 wks after chancre.
- Cardinal signs
    - Skin lesions
    - Mucous patches
    - C. Lata
    - Generalized lymphadenopathy
- Others: Constitutional manifestation

  Liver, Spleen, Bones, Eye, Kidney, CNS, Epididymitis, parotitis (unilateral).

Skin lesions (Syphilides): >80%

-Polymorphic

-Early symm, late asymm

->40% pruritic

-Types:

.Macular (earliest)

.Papular & papulosquamous (commonest)

.Pastular (rare):….Neuro$

NAILS:

Paronychia, onychia= nail loses its luster, becomes brittle, shed

HAIR: follicular syphilides⇨moth-eaten appearance (sides, back of heads+eyebrows)

C. LATA:

Most infectious lesion

Site: moist skin areas

Character:

DD: C.Acuminata

MM: ~30% ⇨ Mucous patches

Sites:tonsils, tongue angle of mouth& nose,larynx

Character: snail track ulcers

DD:……….+hoarseness??

Lymph Nodes: 60% ⇨ Generalized

Site:cervical, occipital, axillary, inguinal, epitrochlear

Character:

DD: HIV, Hodgkin`s Dis, Inf.Mono., Lymphomas, TB

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Macular Syphilide (Roseola)

- The earliest lesion, but being fainty colored ⇨ often overlooked
- Rose pink
- 1 cm
- Discrete
- Indistinct margins
- Pressure obliterates the lesion
- DD: Drug eruption, Measles, Rubella, P. rosea, Inf. mononucleosis

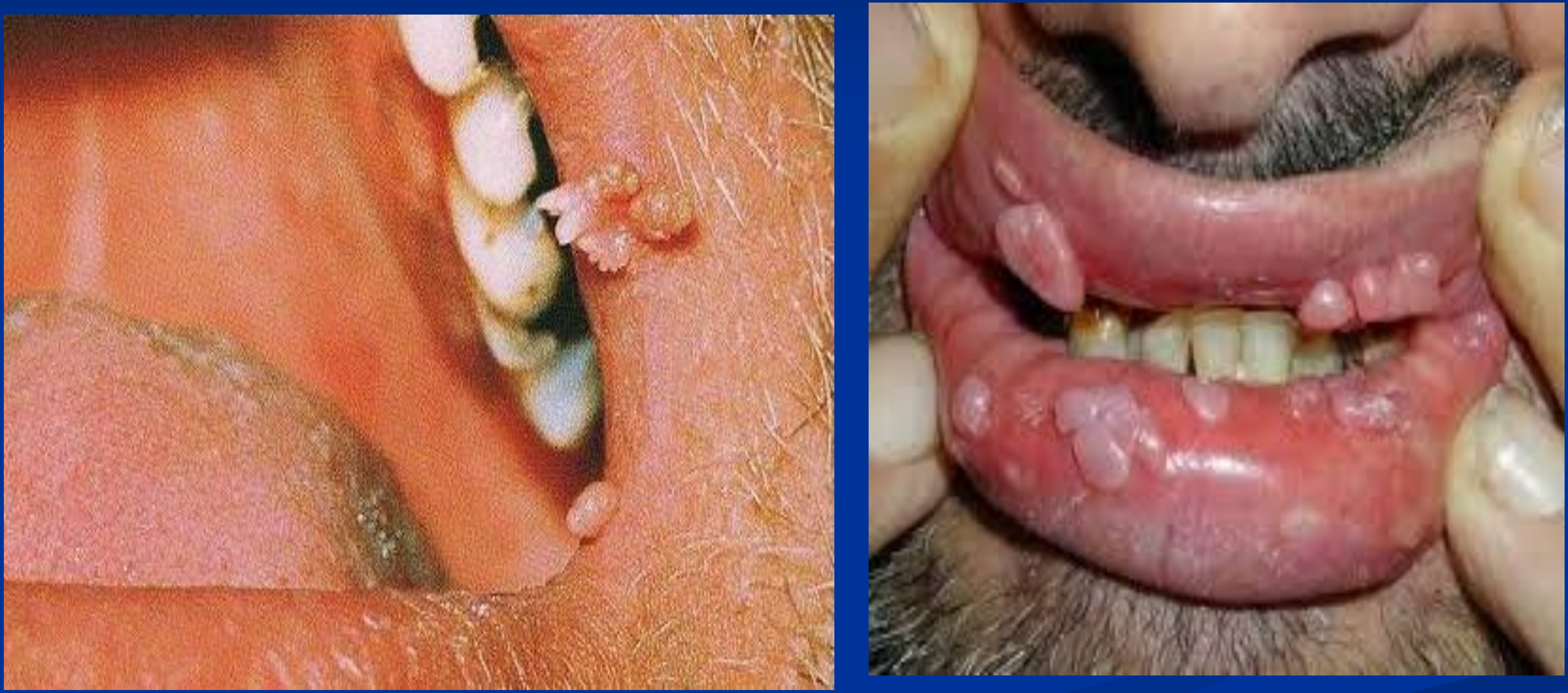
Papular & papulosquamous Syphilide

- The commonest.
- Dull red
- Variable in size
- Firm, shiny surface.
- Symmetrically distributed
- More on Flexor aspects
- With time ⇨ Scaling of `papules ⇨ Papulosquamous
- Site: anywhere on the body, especially: Face, scalp, palms & soles, External genitalia (C.Lata)
- In late stges of 2ry $: papules become fewer, asymmetrical, may coalesce ⇨ Bombshell Syphilide = Large raindrop `e 2ry splashes around.
- DD: Psoriasis, L.Planus, P.lichenoides, HS, Impetigo contagiosa, keratotic eczema, C.acuminata

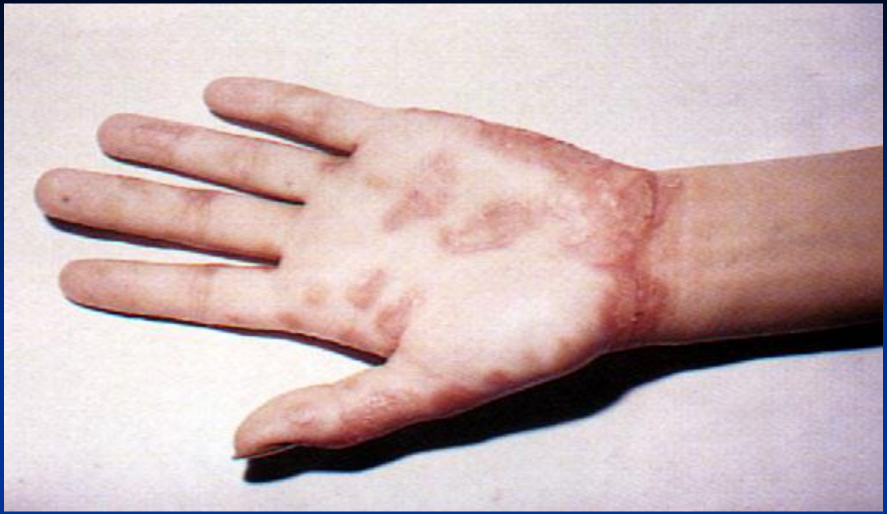
Pustular Syphilide

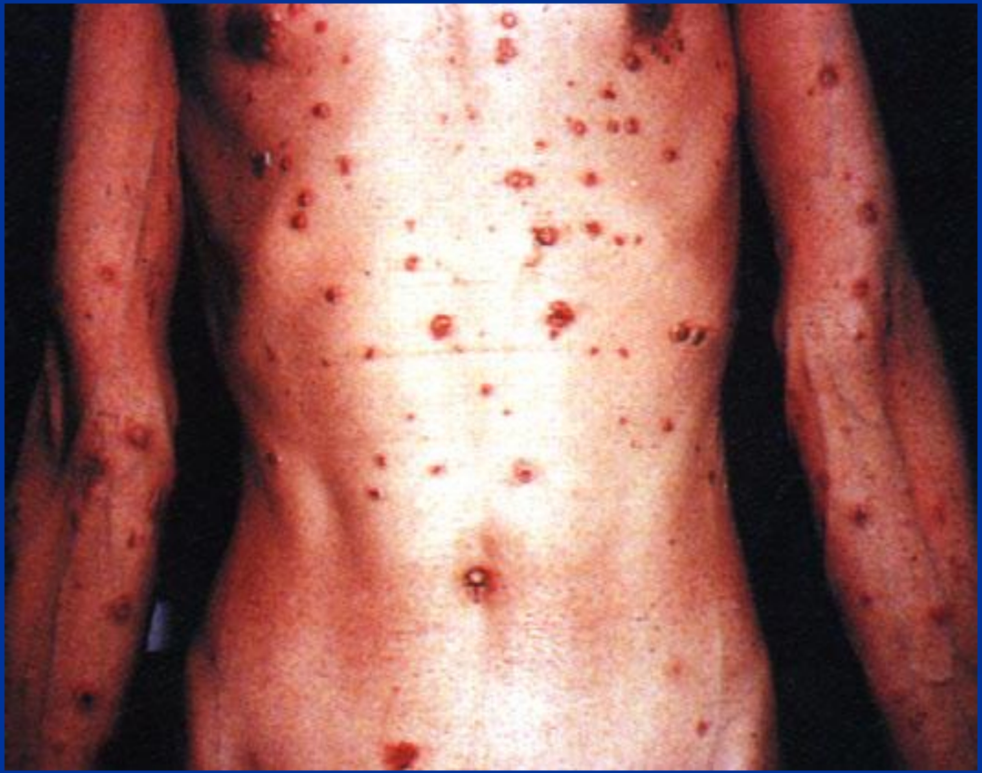
- Rare
- Papule ⇨ necrosis of upper dermis & epidermis ⇨Pustule. With concomitant debilitating diseases (eg TB), immunocompromised.
- DD: Acne Valg., Pyoderma, Scabies, Molluscum Contag., Impetigo.

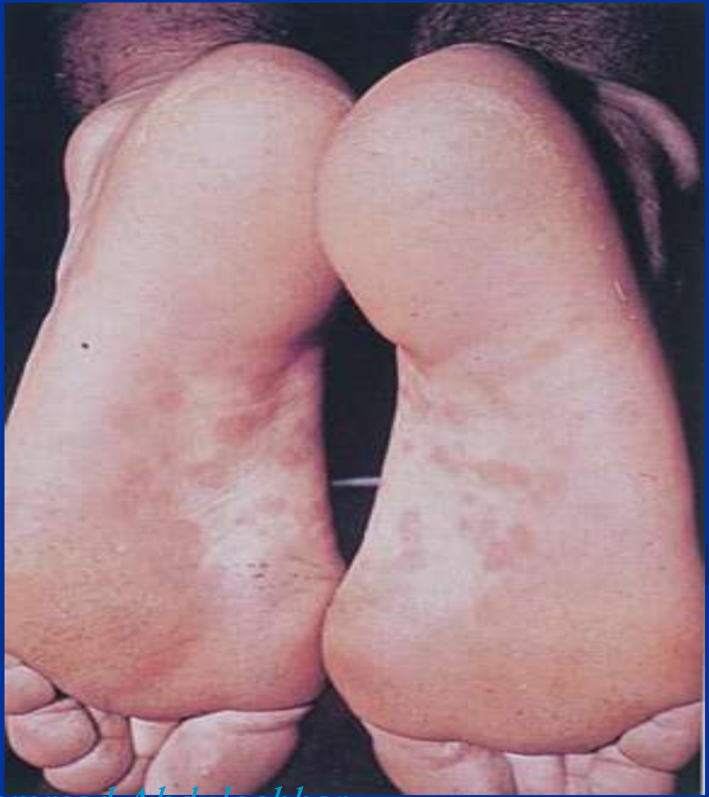
	Condyloma Lata	Condyloma Accuminata
cause	T.P	HPV
Site	Moist skin areas	Genitalia & perianal mainly
shape	Fleshy, sessile, round	Cauliflower, pedunculated
surface	Flat topped	Irregular (mammilated)
color	Dull red	Skin colored
premalignant	no	yes
Lab	+ve DG & VDRL	-ve
Ttt	penicillin	Electro or cryyotherapy, laser, topical podophyllin + others



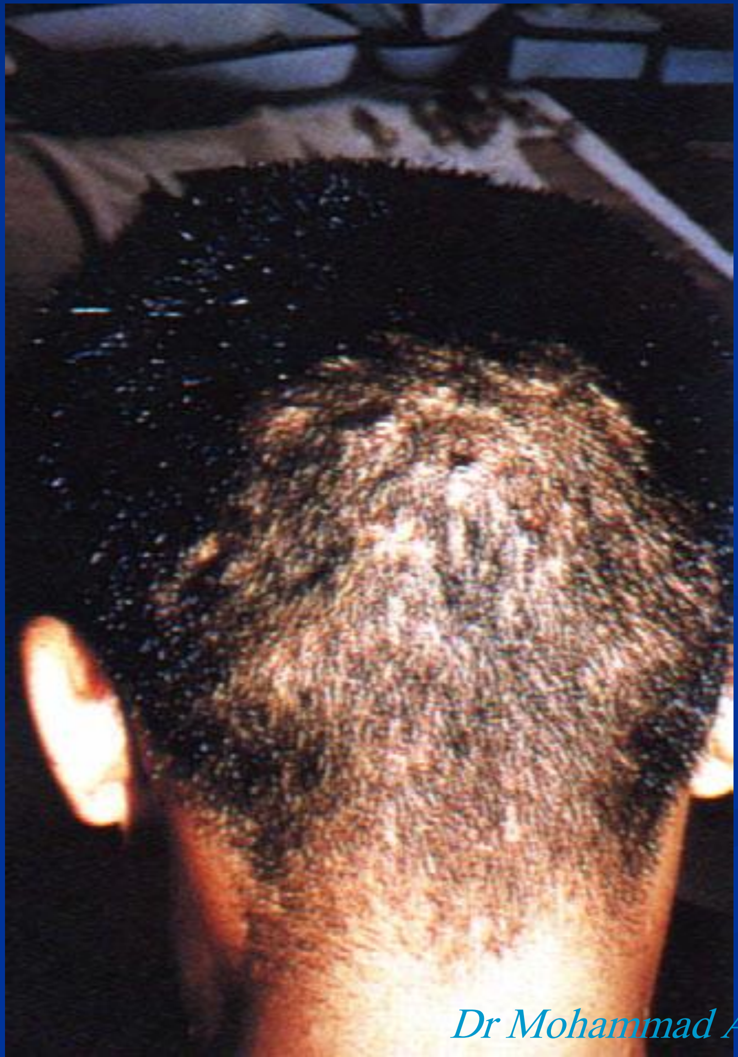
Skin Rash







Syphilitic Alopecia



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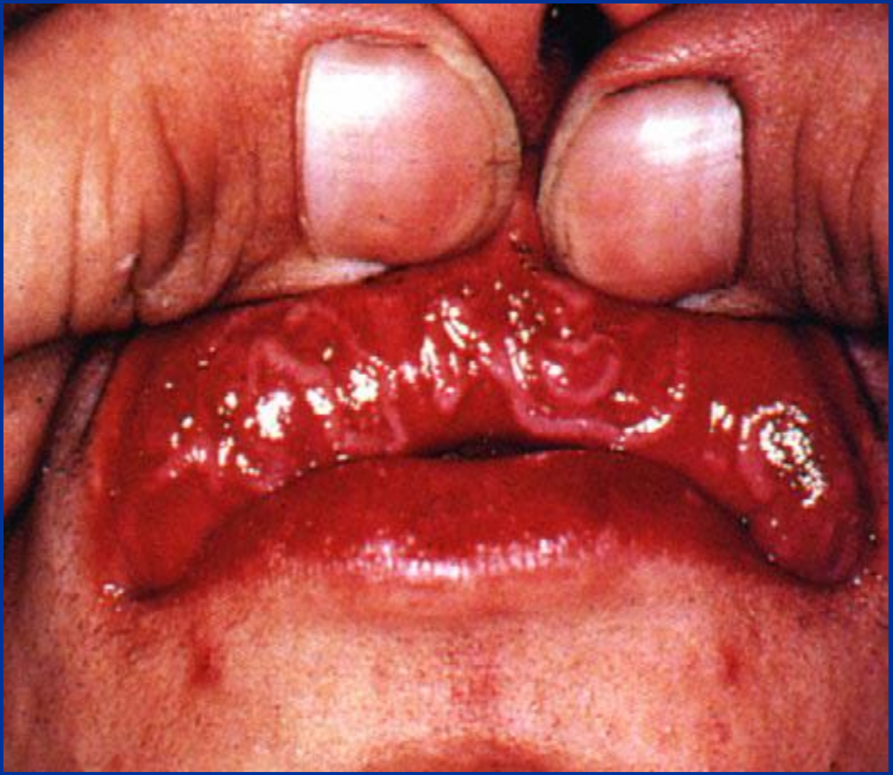
Mucus Patches



Diagnosis of 2ry$

- Darkground Mic
- Serological tests: 100% +ve
- CSF: a MUST to pts `e hearing loss.
- Skin biopsy: $ with HIV

Early latent $

(< 2 years, Infective, Asymptomatic)

- Infective
- Clinical manifestations ⇨ ABSENT
- Serological tests ⇨ POSITIVE

Late Latent $

(> 2 years, Not infective)

- Non infectious. But Preg. MOTHER ???
- Clinical manifestations ⇨ ABSENT.
- Serological tests ⇨ POSITIVE.
- Discovered accidentally
- The danger is the possibility of NEUROLOGICAL or CARDIOVASCULAR Complications

Benign tertiary: (Gumma)

=central necrosis surrounded by zone of granulation tissue (plasma cells & lymphocytes) surrounded by narrow zone of fibrous tissue

- Skin & M.M.
- SC tissue.
- Bones & cartilages.
- Viscera e.g. testis, liver, stomach

Gumma of Skin

- Nodules
- Single or grouped
- Not symmetrical
- Rounded, red, freely mobile
- May heal by thin scar or pigmentation
- Gummatous ulcer: Clean surface & punched out edge. Regional LNs= not enlarged
- Sometimes on palms & soles (psoriasiform gumma)

Gumma of SC tissue

- Painless, single or multiple lumps
- Dull red
- Later attached to overlying skin= Ulcer
- Main sites: lower legs, face, & buttocks.

Gumma of MM

- Palate ⇨ perforation
- Nasal septum ⇨ destruction
- Larynx ⇨ stenosis
- Tongue ⇨ 2 forms

    1) Localized ⇨ ulcer

    2) Diffuse ⇨ diffuse interstitial fibrosis (chronic superficial glossitis) , leukoplakia…& Squamous CA.

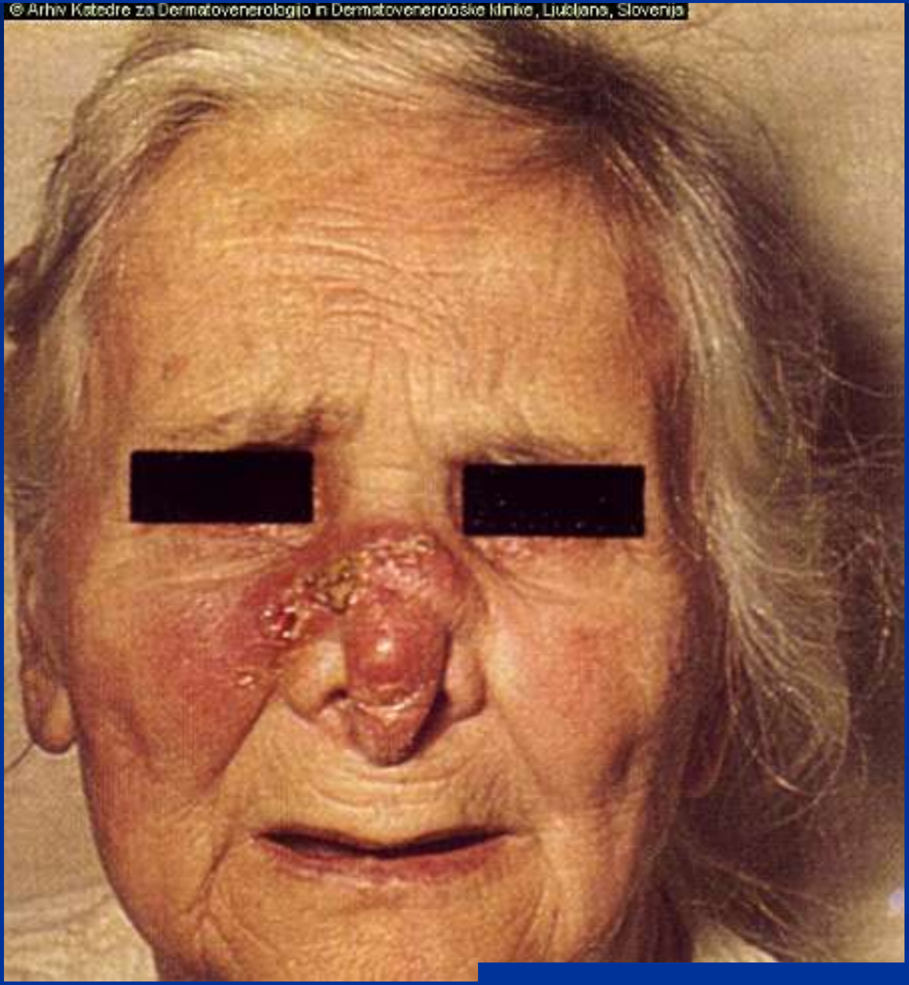
Gumma of Bones

- **Long bones**: Syphylitic periostitis (eg; tibia)…new bone formation.
- **Flat bones**: Syphylitic osteomyelitis (eg; skull)….no new bone formation (bone destruction due to affection of medullary cavity)

Gumma of Viscera (Localized or Diffused)

- Liver
- Stomach
- Testes

Gumma



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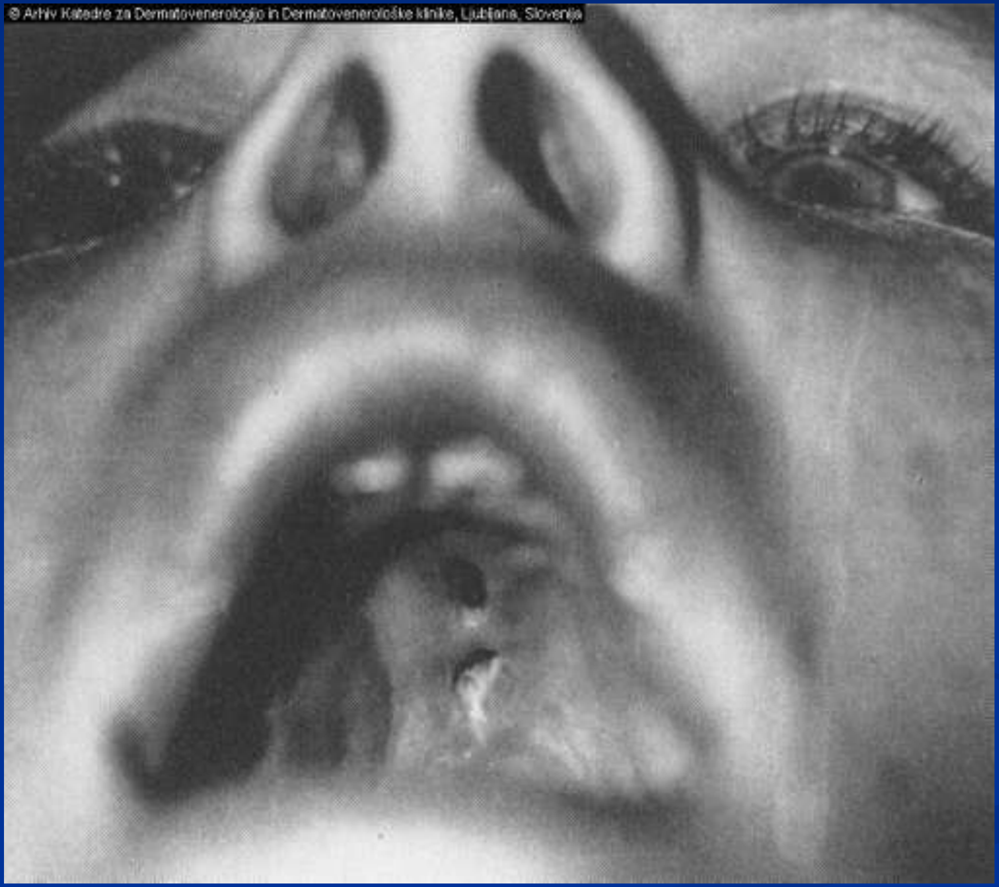
Gumma of Palate



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Malignant tertiary

- **Cardiovascular $**:
  - $ of the heart: Localized or diffuse
  - $ of the medium vessels: cerebral & spinal , carotid, iliac & femoral arteries.
  - $ of great vessels: Asymptomatic, aortic regurge, aortic aneurysm or coronary osteal stenosis
- **Neurosyphilis**:
  - Asymptomatic
  - $ of brain:
    - Meninges
    - Blood vessels
    - Parynchyma (general paralysis of insane
  - $ of spinal cord
    - Meninges
    - Blood vessels
    - Parynchyma (Tabes dorsalis)

Congenital Syphilis

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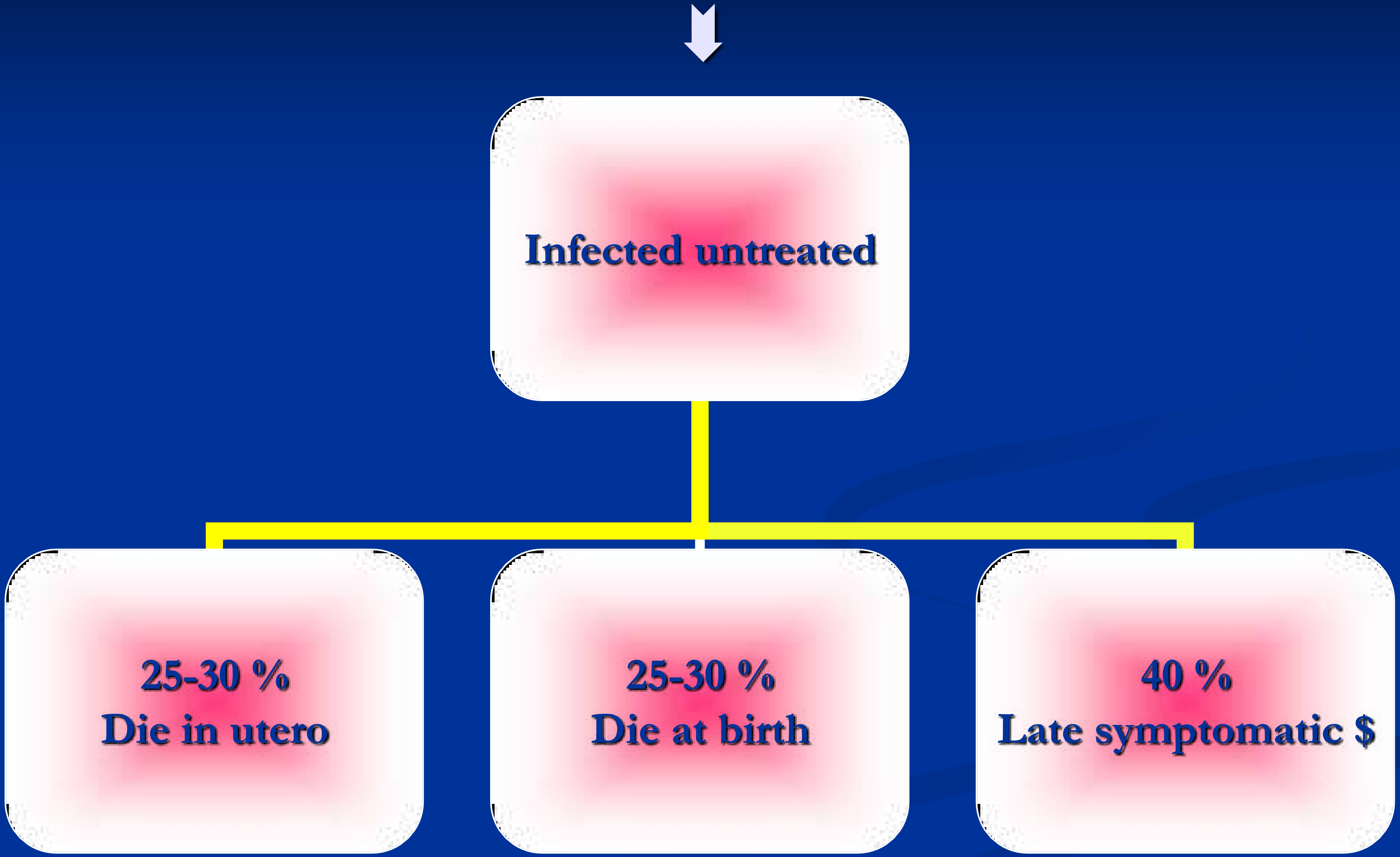
Preg. If treated ⇨ 14% deliver stillbirth or infected. If not treated 80-90%



Infected untreated

25-30 %
Die in utero

25-30 %
Die at birth

40 %
Late symptomatic $

Congenital syphilis

T.P. pass placenta after 4th month ⇨

Kassowitz law:

- Abortion after 4th m of preg.
- Premature
- Stillborn
- Live born ⇨ signs of $.
- Live born ⇨ remains healthy.

Healthy baby at birth ⇨ **Profetal law**

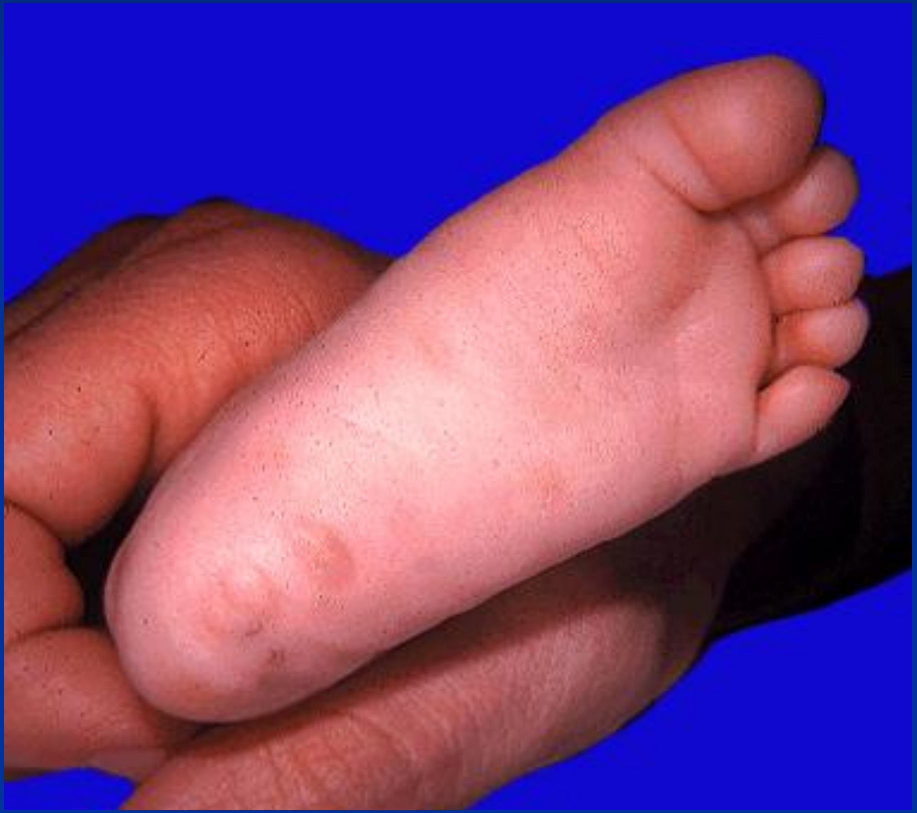
- Bl. From umbilical vein may give –ve serology ⇨ does not exclude $, **WHY???** Follow-up during 3 months.
- Bl. From umbilical vein may give +ve serology ⇨ does not prove $, **WHY???** **IgG**

Congenital Syphilis

Early Congenital (1^{st} 2 years =2ry $):

- Skin rash (??Bullous lesions in palms & soles)
- Mucous patches & Condyloma Lata
- Generalized lymphadenopathy
- Constitutional symptoms
- Eye manifestations
- Others: Hepatitis, osteoperiostitis, periostitis, osteochondritis, meningitis,….etc

Skin Rash



Late Congenital $ (after the first 2 ys= tertiary $):

- Gumma (skin, joints, bones, viscera)
- Interstitial keratitis
- 8th nerve deafness
- Clutton's joint
- Neuro $
- CV $ (rare)

Stigmata of Congenital Syphilis

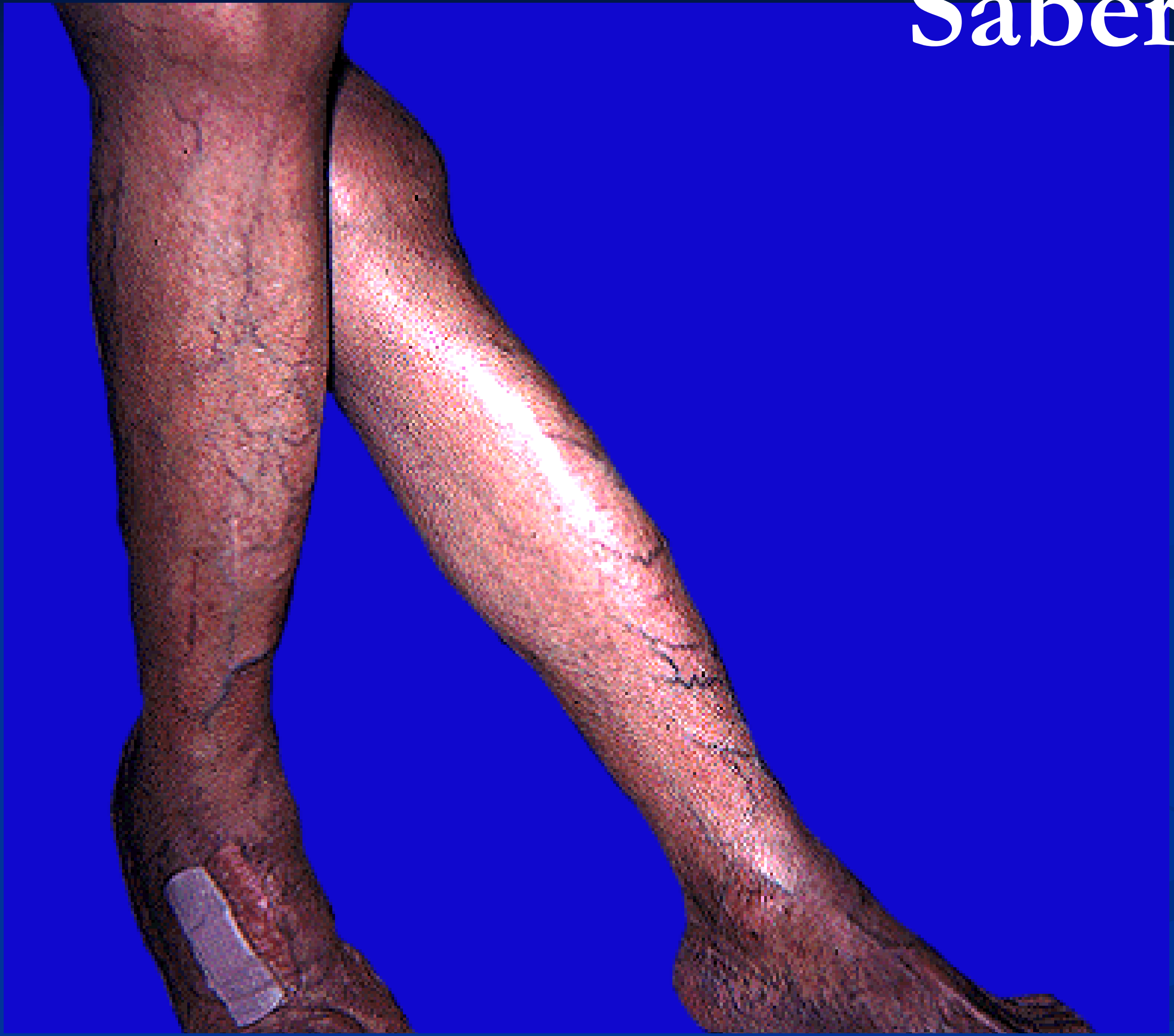
Stigmata of early $:

- chorioretinitis
- Hutchison's teeth
- Moon's molars
- Rhagades
- Saddle nose.
- high arched palate
- bulldog face

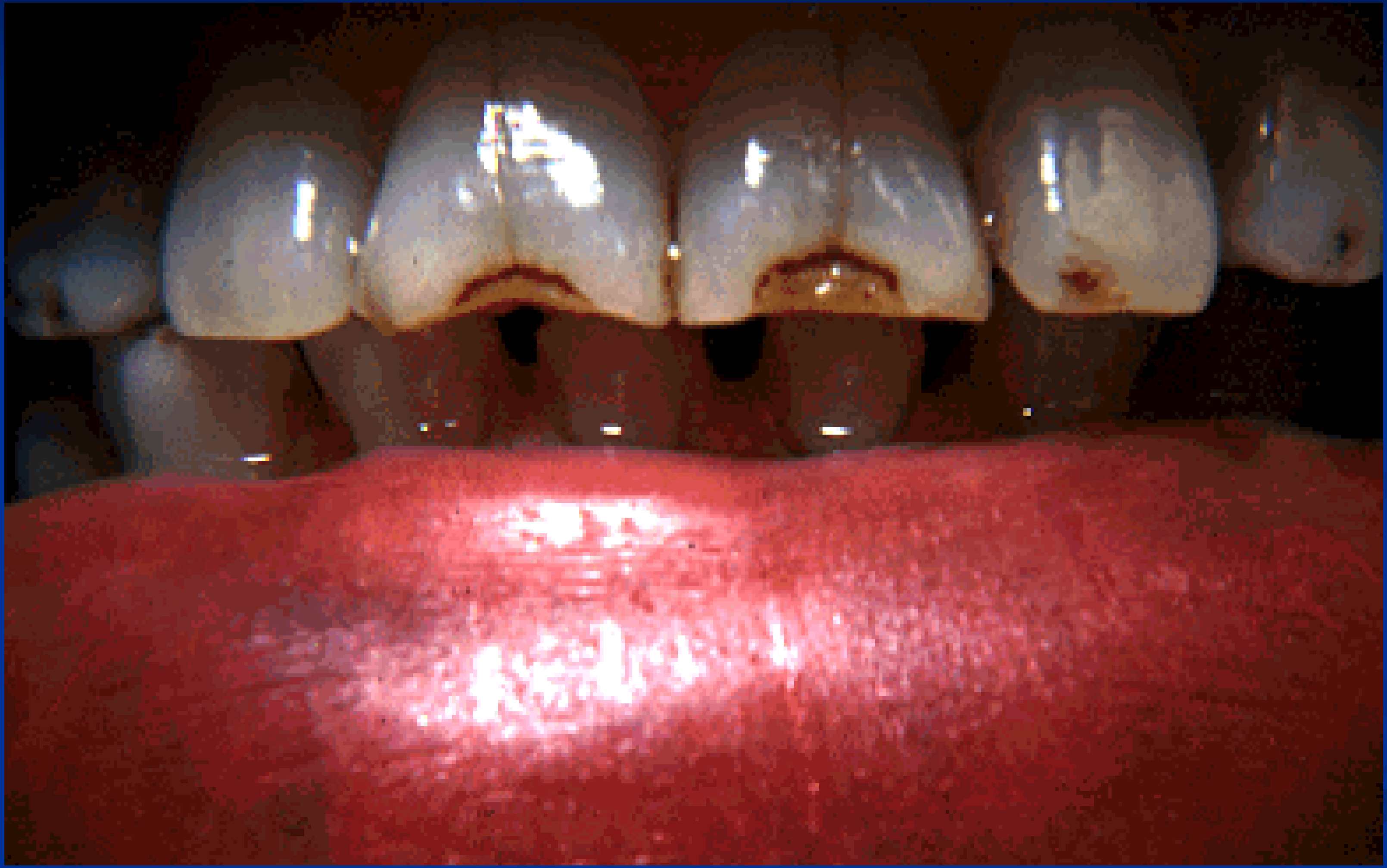
Stigmata of late $:

- Corneal opacities, optic atrophy
- Perceptive deafness
- Sabre tibia
- Frontal bossing of the skull.

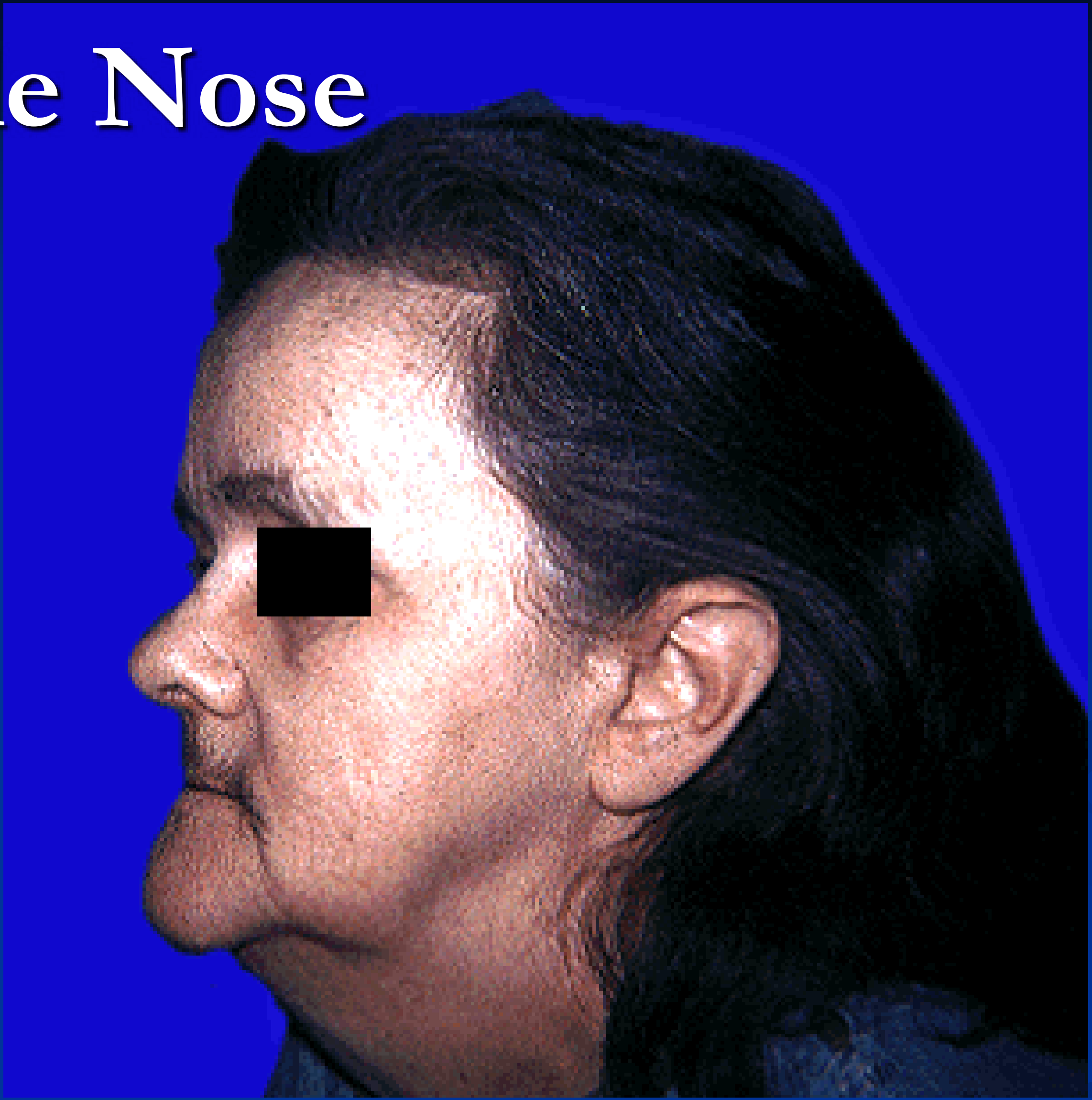
Saber Tibia



Hutchison's teeth



Saddle Nose



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Hutchinson triad

1) Interstitial keratitis
2) Peceptive deafness (8th nerve)
3) Hutchinson teeth

Investigations

A. **Dark Ground Examination**

B. **Serological tests**

  1) **Non-specific (Reagin antibodies / cardiolipin):**

    a) **Flocculation tests**

    - VDRL (Venereal Disease Research Laboratory) Test
    - RPR (Rapid Plasma Reagin)

    b) **Complement fixation test**: ⇨ Wasserman reaction

  2) **Specific (treponemal antigens):**

    - FTA (Fluorescent Treponema Antibody test)
    - FTA-absorbed Test
    - TPHA test (Treponema Pallidum Haemagglutination)
    - TPI (Treponema Pallidum Immobilization)

Serological tests

I- Non-specific tests:

- Idea
- Antigen ➪ Cardiolipin
- Reagin ➪ Antibodies in $ serum
- Types: Flocculation, Complement fixation
- Advantages (Cheap, Simple, Screening, Follow up)
- Disadvantages (False +ve, False –ve, Biological false +ve reactions).

False +ve reaction

Acute

-Acute febrile infections (Chicken pox, measles, malaria, hepatitis, IM, viral pneumonia)

-Strong immunological Stmuli (Prg., Vaccination)

Chronic

-CT diseases

-Leprosy

-TB

-Malignancy

-Autoimmune (Hemolytic a, thyroiditis…….)

False –ve

⬇

Prozone phenomenon due to:

High conc. of Abs in pt. serum

II- Specific tests

- Idea
- Antigen ⇨ treponemal antigen
- Antibodies ⇨ Antibodies in $ serum
- Advantages (Accurate, Confirmatory, no false results)
- Disadvantages (Expensive, difficult tech., not used for follow up ⇨ always +ve)
- Types: TPHA, TPI, FTA, FTA-Abs, RP-CFT

Treatment

- Early Stage
- Cardiovascular $
- Congenital $

- Late Stage
- Neuro $

- ❖Pregnancy
- ❖Allergy
- ❖HIV

- Sex partner
- Missing a dose
- Follow up

- To penicillin
- To procain
- Jarisch-Herxheimer

Early $:

Adult:

- **Benzathine penicillin G** 2.4 million units IM in a single dose. OR
- **Procaine Penicillin:** 600,000 U, IM daily ⇨ 10ds

(**Multiple injections)**

Child:

Benzathine penicillin G 50,000 units/kg IM, up to the adult dose of 2.4 million units in a single dose.

Late $ or unknown duration:

Adult

- ❑ **Benzathine penicillin G** 2.4 million units IM /week for 3 weeks. OR
- ❑ **Procaine Penicillin:** 600,000 U, IM daily ⇨ 20ds

Child: **Benzathine penicillin G** 50,000 units/kg IM, up to the adult dose of 2.4 million units/wk for 3 wks.

Cardiovascular $ = Late stage + specific for HF or aneurysm.

Neurosyphilis = Not **Benzathine penicillin**, **WHY?**

A) **Recommended**:

-**Crystalline penicillin G:** 3-4 million units **IV** every 4 hours or continuous infusion, for 10-14 days.

B) **Alternative**:

-**Procaine penicillin** 2.4 million units IM once daily

PLUS

-**Probenecid** 500 mg orally four times a day, both for 10-14 days.

Allergy: ceftriaxone 2g/d IM,IV ⇨ 10-14ds

Child: **Crystalline penicillin 50,000 U/Kg daily for 15ds.**

Allergy to penicillins
(close follow-up is essential)

- Tetracycline: 500mg X 4 X 14ds in early $, & 28ds in late $, empty stomach
- Doxycycline: 100mg X 2 X 14ds in early $, & 28ds in late $.
- Ceftriaxone: 1g/d (IM/IV) for 8-10ds (test for allergy).

Pregnant

SAME according to stage

Follow up / month quantitatively until delivery.

Allergic: Desensitization

Syphilis with HIV

As Neurosyphilis

Reactions to Treatment

- To Penicillin:
  - Immediate anaphylactic reaction ⇨ fatal
  - Delayed urticarial reaction ⇨ mild
- To Procaine: V. rare ⇨ neuropsychiatric manif.
- Vasovagal attack
- **Jarisch-Herxheimer:**

Jarisch-Herxheimer:

- Following the 1st dose. Within 24 hrs of ttt.
- Caused by waste products of destructed T.P ⇨ immunological & hypersensitivity reaction.
- Acute febrile reaction + headache & myalgia. In 50 % with early $, & 25 % of late $.
- Intensification of local or systemic manifestation
- May cause early labor or fetal distress in preg.
- Ttt: oral prednisone

Follow up

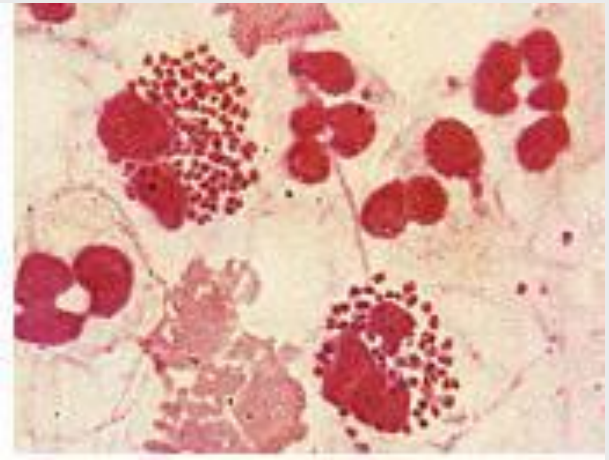
- By quantitative non-treponemal serological tests.
- 6, 12, 24 months
- Titre should be decreasing by 4 folds from baseline.
- Re-treatment is recommended if:
    - Titres > 4 folds
    - An initially high titre (> or = 1:32) fails to decline at least 4 folds within 12-24 ms of therapy
    - Signs or symptoms of syphilis develop

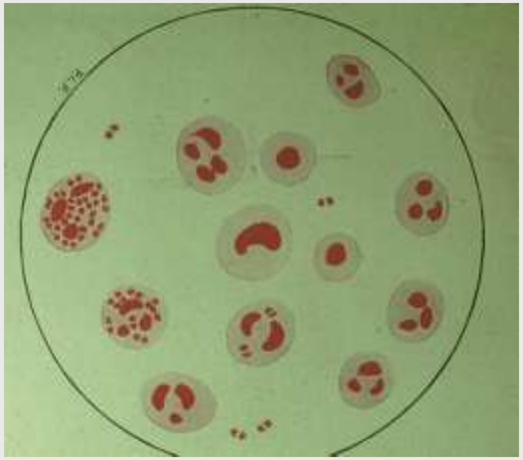
Gonorrhea

Causative Organism

Neisseria gonorrhea

kidney shaped, gram –ve diplococci (in pairs), intra and extracellular.





Mode of Transmission:

Sexual: Homo- or heterosexual relations, orogenital

Asexual: -Contaminated towels or hands (less common)

-Vertical transmission: ophthalmia neonatorum

Virulence factors: Pili, LOS (endotoxin), Proteases (inhibition of IgA secretion)

Affinity to columnar epithelium

Pathological effect

- Patch destruction of mucosa= Burning micturition and painful symptoms
- PNLs in submucosa= PUS
- Fibrous tissue formation= Obstructive infertility

Sites of involvment

- Males= urethra, Littre’s, cowper’s glands, prostate, Svs, Epididymis, rectum

Clinical Picture:

- IP: 2-7ds (maybe more)
- Genital affection in **males**
- Genital affection in **females**
- **Extra-genital** gonorrhea
- Gonorrhea in **infants** & **children**
- Gonorrhea in **pregnancy**
- **Disseminated** gonorrhea

Genital affection in males

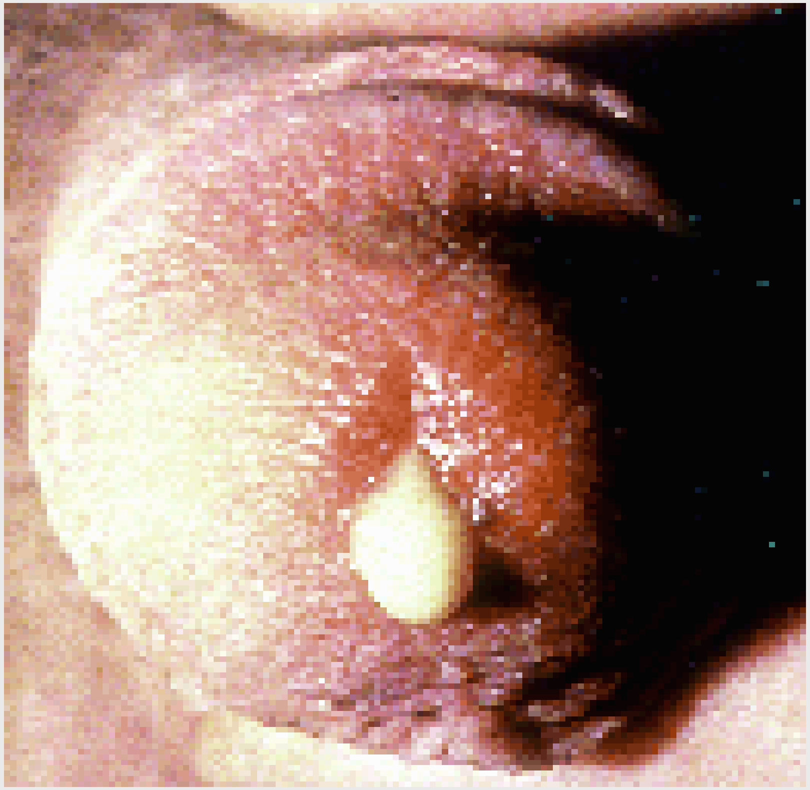
Symptoms & signs:

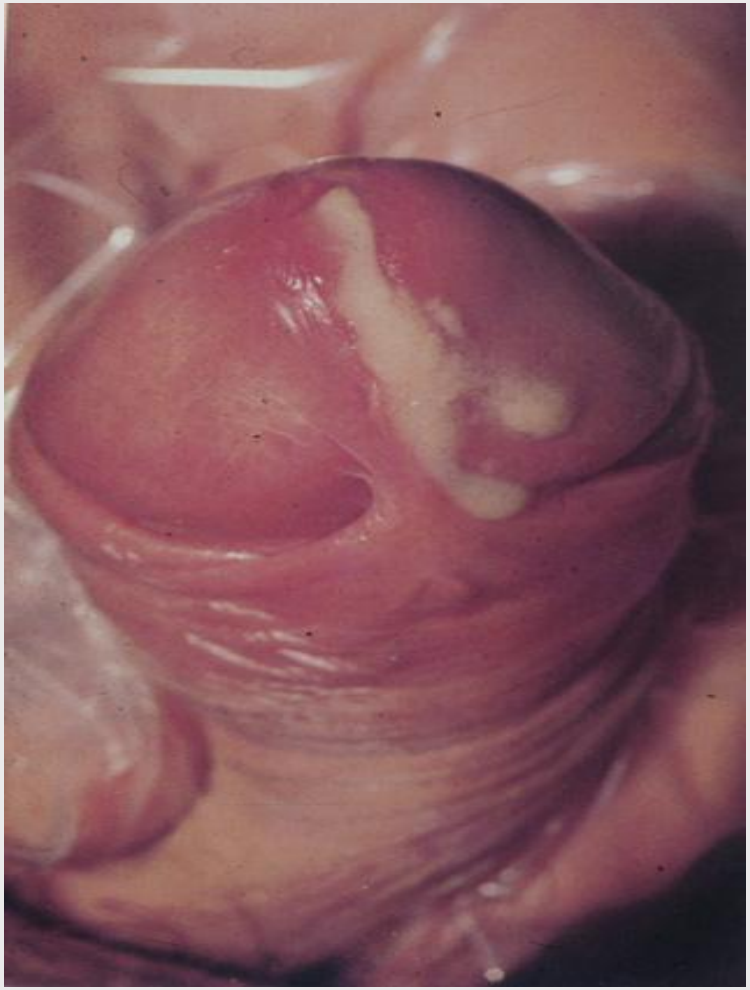
- Asymptomatic (<15%).
- Urethral discharge: profuse, purulent, yellowish
- Urinary symptoms: dysuria, frequency, urgency and hematuria (posterior urethritis).
- Hyperemia and oedema of urethral orifice
- Ing. LNs ??.

Complications (local, systemic):

- Balano-posthitis, peri-urethral abscess & fistula, urethral stricture
- Tysonitis, Littritis, Cowperitis
- Acute prostatitis, seminal vesiculitis and epididymitis

Urethral Discharge





Genital affection in females

Symptoms & signs:

- Asymptomatic (50%)
- Urethral meatus, cervix, LNs
- Genital (not vaginal) discharge: mucopurulent
- Urinary symptoms: dysuria, frequency, urgency and hematuria
- Low back pain or lower abdominal pain

Complications (local, systemic):

- Cervical gland affection (Nabothian follicles), Skenitis, Bartholinitis & Bartholine abscess.
- Salpingitis, oophoritis, tubo-ovarian abscess & pelvic peritonitis.
- Pelvic inflammatory disease

Extra-genital gonorrhea

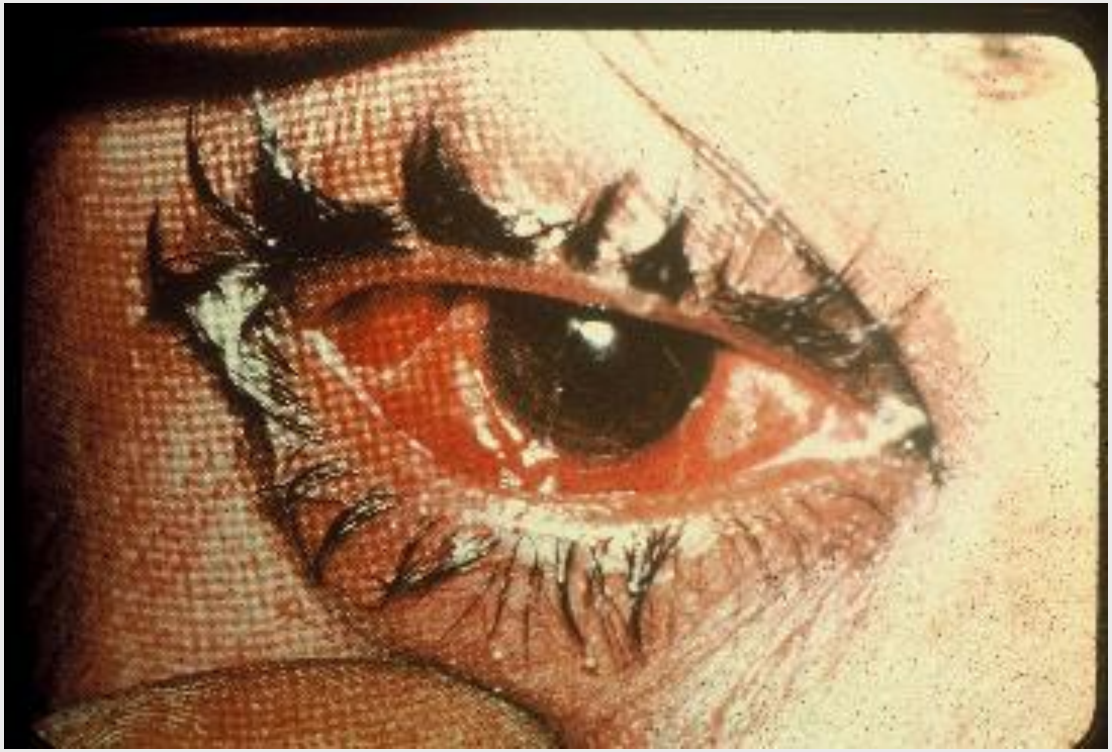
- Proctitis
- Pharyngitis (orogenital contact)
- Conjunctivitis (direct contamination)

Disseminated gonococcal infection

2-3 weeks of 1ry infection

- Dermatitis (maculopapular or pustular acral lesions), extremities (around joints) > trunk.
- Arthritis (migrating polyarticular, supurative, large joints; knees, hands, feet). Purulent joint fluid.
- Perihepatitis (Fitz-Hugh-Curtis syndrome).
- Iridocyclitis (blood spread).
- Meningitis, endocarditis.

Gonococcal conjunctivitis



Gonorrhea in infants & children

Ophthalmia neonatorum:

- Acquired during vaginal delivery from gonococcal cervicitis.
- Occurs within 2-5ds after delivery
- Manifestations: purulent eye discharge with edema of eyelids, preauricular LN enlargement. Globe perforation and blindness. CNS???
- Management (**TEST FOR C. TRACHOMATIS**):

- Ophthalmia neonatorum prophylaxis: Erythromycin 0.5% oint, or tetracycline 1% oint single use to both eyes (delivered Vag. Or CS).

- Systemic: Ceftriaxone 25–50 mg/kg IV or IM in a **single dose**, not to exceed 125 mg. No need for topical ttt.

- Follow up: MUST.

- Don`t forget to treat the mother and her partner.

Gonococcal vulvo-vaginitis

- Infection occurs through Sexual abuse, contaminated towels or toilet seats.
- **Clinically**, there is vulval pruritus, vaginal discharge and burning micturition, proctitis.
- **Recommended treatment:**
  - For Children Who Weigh **>45 kg** = adult regimen.
  - For Children Who Weigh **<45 kg** and Who Have Uncomplicated Gonococcal Vulvovaginitis, Cervicitis, Urethritis, Pharyngitis, or Proctitis = Ceftriaxone 125 mg IM in a single dose.
  - For Children Who Have **Bacteremia or Arthritis** = Ceftriaxone 50 mg/kg (maximum dose: 1 g) IM or IV in a single dose daily for 7 days.

All children who have gonococcal infections should be evaluated for coinfection with **syphilis** and **C. trachomatis.**

Gonorrhea in Pregnant females

- Pregnancy is not a special risk factor
- Disseminated gonococcal infection is more common in pregnancy.
- Salpingitis occurs early.
- Chorioamnionitis, spontanous abortion, preterm rupture of membranes, low birth weight may occur.
- Post-partum, post-abortion sepsis
- **Treatment** :
  - No quinolones or tetracycline
  - Treat with Cephalosporin or single 2-g dose of spectinomycin IM.
  - Presumptive chlamydia inf.: azithromycin 1g single dose OR
    amoxicillin 500mg 4x for 7ds.

Lab Diagnosis of Gonorrhea

1 - Stained smear:

- **Gram stain (**kidney shaped, gram –ve diplococci (in pairs), inside and outside the PMNLs.
- **Immunoflurescent stain**

2 – Culture (WHEN??):

- **Growth Media, selective (Modified Thayer-Martin medium)**
- **Trans-Growth Media**

3 - Colonies confirmatory tests:

-Gram stained smear examination from colonies.

-Oxidase test: Oxidase reagent turns gonococcal colonies (in mixed cultures) black.

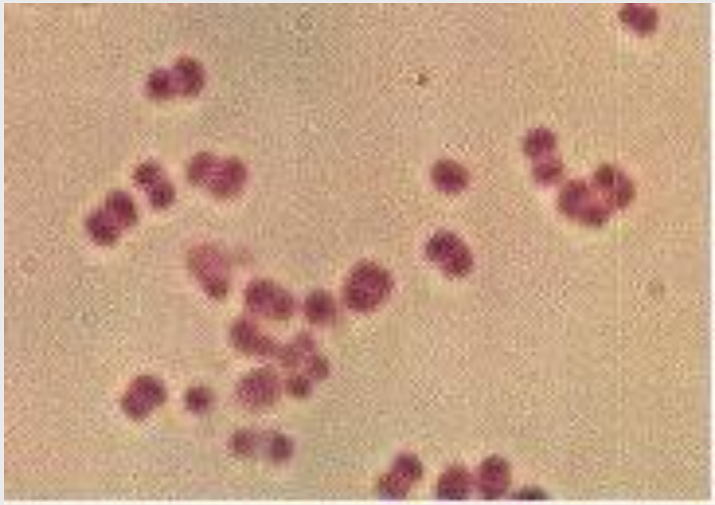
Sugar fermentation test (gonococcus ferments glucose only).

4- Auxotyping (AUH).

5- Serological tests (ELISA, RIA, PCR); Antigen, Antibodies, and Gene detection

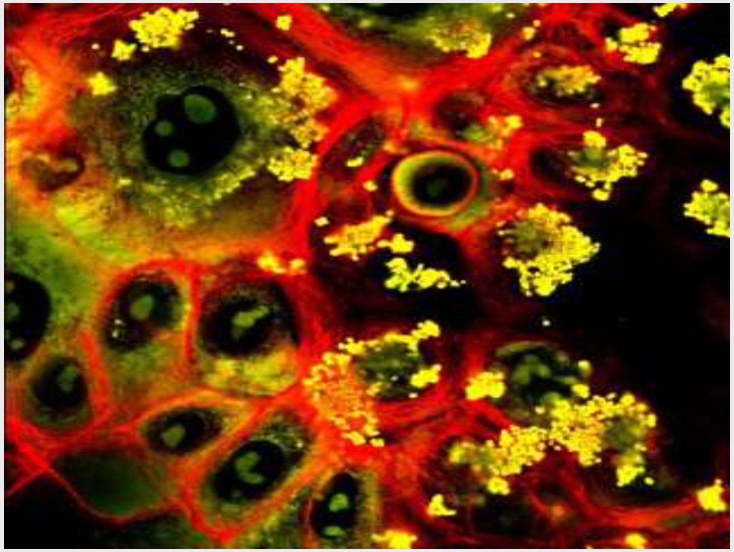
- The high **sensitivity** and **specificity** of the combination of **Gram stain** and the **Culture** make the routine use of other tests unnecessary and indicated only in selected cases

- Asymptomatic male
- Chronic cases.
- Females
- Rectal or oro-pharangeal infections.
- Disseminated infection (Detection of antibiotic sensitivity).
- Medico-legal.



Gram stained smear

Immunoflurescent stained smear



Colonies of *N. gonorrhoeae*

Gonococcal Cultrue

TREATMENT

- Prevention and control.
- Treatment of the disease
- Treatment of associated diseases
- Treatment of the complications
- Treatment of sex partner (within 60 ds or if >60ds, the most recent one)
- Follow up

- Prevention of:
  - Reinfection
  - Spread of infection
  - Traumatic urethritis
  - Chemical urethritis

Uncomplicated Gonococcal Infections of the Cervix, Urethra, Rectum and Pharynx + Conjunctivitis (+saline)

Ceftriaxone 250mg IM in a single dose

PLUS

Azithromycin 1 g orally in a single dose

Alternative (if ceftriaxone is not available)

Cefixime 400 mg orally in a single dose

PLUS

Azithromycin 1 g orally in a single dose

Alternative Regimens

- Spectinomycin 2 g in a single IM dose

OR

- Single-dose cephalosporin regimens

OR

- Single-dose quinolone regimens

Treatment of complicated cases

Disseminated Gonococcal Infection

- **Arthritis and Arthritis-dermatitis syndrome**

  Ceftriaxone 1g IM or IV/ 24hrs

  PLUS

  Azithromycin 1 g orally in a single dose

Alternative

Cefotaxime 1 g IV every 8 hours

PLUS

Azithromycin 1 g orally in a single dose

Gonococcal Meningitis and Endocarditis

Ceftriaxone 1–2 g IV every 12 hours

PLUS

Azithromycin 1 g orally in a single dose

- Therapy for **meningitis** should be continued for 10–14 days; therapy for **endocarditis** should be continued for at least 4 weeks..

Thank You